









**HUTCHISON'S  
CLINICAL METHODS**

### *Historical Note*

SIR ROBERT HUTCHISON wrote the greater part of *Clinical Methods* at the age of twenty-five, when he was a resident at Great Ormond Street. The remainder was written by Dr Harry Rainy, University Tutor in Clinical Medicine at Edinburgh. The book was published in 1897, shortly after Hutchison had been made Demonstrator in Physiology in the London Hospital Medical College, and three years before he was appointed to the staffs of the London and Great Ormond Street Hospitals. He died in the fullness of years, wisdom and honours on January 12th, 1960.

### *From the Preface to the First Edition*

THE title *Clinical Methods* probably describes the scope of this book better than any other. It is not intended as a treatise upon medical diagnosis. On that subject there is already a sufficiency of good works in existence. It aims rather at describing those methods of clinical investigation by the proper application of which a correct diagnosis can alone be arrived at. To every student when he first begins work in a medical ward the question presents itself: How shall I investigate this case? To that question the present work is intended to provide an answer. The first chapter deals, therefore, with the methods of case-taking in general, and includes a general scheme for the investigation of medical cases. The rest of the book is really an expansion of that scheme, each system being taken up separately, and the methods of investigating it described in detail.

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FIFTEENTH EDITION

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## PREFACE

*Clinical Methods*, unlike its authors, grows slimmer with the passing of the years. This is mainly due to a change of format, but in part to the pruning of a lot of redundant material.

The book has undergone a complete and thorough revision, in which we have once more had the valuable assistance of many of our colleagues at the London Hospital. Dr David Penington has been responsible for a part of the revision and has written a new chapter on the clinical assessment of metabolic disorders. Mr Charles Keogh has written a new section on hearing and tests of auditory and vestibular function. Mr Barrie Jay has rewritten the section on the examination of the eye and Dr Anthony Jackson that on the examination of children. Dr John Perrin has provided us with new colour photographs of blood and bone marrow and Professor G. R. Seward with new photographs of the tongue. We have also had assistance from Dr Harry May, Dr J. S. Pegum, Dr J. F. Robinson, Dr D. T. Hughes, Dr J. D. Ward, Dr H. L. F. Currey, Dr J. Hamblin and Dr R. B. Pridie; and to each and all of these kind friends we acknowledge our indebtedness.

With the star or spectre – as you will – of the computer hanging over us, the student might be led to think that the importance of clinical method is receding. There is a saying in the computer world – ‘garbage in, garbage out’. If a machine is really going to suggest diagnoses to us, it will only do so usefully if it is first fed with accurate information suitably standardized and coded. Accurate information about the history and physical examination still depends on clinical method. We suggest therefore that good clinical method, far from receding in importance, will be quite as necessary in the future as it always has been, and we venture to hope that *Clinical Methods*, now past its seventieth year, will continue to be of assistance both to students and postgraduates in the years to come.

D.H.  
R.R.B.



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# **1 CASE-TAKING**

There can be no question about the value of accurate and systematic case-taking, but about the method to be pursued there can be differences of opinion. The method should be comprehensive so as to be capable of being applied to every case and to cover all relevant points. The resulting case notes should be concise, yet present all the important features of the case. Conciseness is of great importance. Nothing is more annoying than to have to wade through a mass of verbiage to get at the chief facts of a case. The student should practise the art of presenting the leading features in a few sentences. He should avoid verbose descriptions. Simple outline drawings can often convey information that would otherwise require many sentences.

The scheme of case-taking appended to this chapter must be used with judgement and elasticity. All the points mentioned need not be inquired into in each individual case. For example, if a patient is suffering from advanced heart disease, it is no use writing a minute description of the state of his teeth. Some experience is required to assess what special inquiries are important in particular cases, but common-sense will prevent gross blunders.

The 'taking' of a case consists of two parts:

I The interrogation of the patient—the 'history'.

II The physical examination.

Clinical clerks will find it best to make rough notes on both of these, and afterwards to write out the whole case in detail.

## **I THE INTERROGATION OF THE PATIENT**

The object of interrogation is to obtain information about the patient's present illness, his previous health, and that of his family. The interrogation must be patiently carried out, the patient being allowed, as far as possible, to tell his story in his own words. One patient is a good witness and another poor. Some give an excellent history. Some seem quite unable to give any precise account of what

they feel to be wrong. This may be due to stupidity or to the effects of disease on their mental faculties. Some, however, fail to understand the need for accurate information, and feel that if only they can impress the doctor with the urgency of their distress (which in such cases often turns out to be emotional in nature) he will be able as if by magic to relieve them of it. It is important to recognize the reason for the evasiveness of such patients and not to allow oneself to become annoyed with them. The manner of questioning may make all the difference in the world to a nervous or suspicious patient. One may defeat one's own ends by wounding the sentiments or conscience of the patient long before the physical examination begins. It is also important to avoid asking the same question twice, because this conveys to the patient the impression that one is not taking a real interest in his case.

All symptoms are not of equal diagnostic importance. There is usually one symptom which troubles the patient more than any other, and which can be described as the *presenting symptom*. Special attention should always be given to it. It is a good rule not to ask leading questions when taking the history, but once a tentative diagnosis has been made on the presenting symptom it is well to ask for corroborating symptoms, which the patient may not have specially observed or which he may have forgotten to mention. The experienced doctor shows great skill in the choice and wording of these leading questions. The student will learn this art by experience.

### 1 The General Interrogation

Begin by ascertaining the patient's name, age, occupation, and whether he is married or single. It is also important to note his exact postal address.

Two important questions then follow—(1) What does he complain of? To ask 'What is the matter?' is a mistake, as this invites the retort that that is what the patient has come to find out. (2) How long have the symptoms been present? The patient usually dates the start of his illness from some impressive event or from the onset of some severe symptom. Questioning will sometimes reveal earlier symptoms which belong to the history of the present illness. The real date of onset can often be more accurately defined by asking such questions as 'When were you last quite well?' Sometimes it is useful to ask 'Did you ever have a pain like this before?' Having thus defined his complaint and its duration, go on to find out the chief facts in his history.

The most logical plan is to take the *family history* first, but in

practice it is perhaps more convenient to begin with the history of the present illness, to pass from that to an inquiry into the patient's previous health, and go on to the family history. It is usually sufficient to inquire about the state of health or cause of death of the immediate relatives only—of the parents, brothers and sisters, and, if the patient is married, of his own children, if any. These facts may tell us whether he is predisposed by heredity to any particular disease, or whether he has reason to be anxious about a particular disease because he has seen a relative suffer and perhaps die from it.

Next comes the *social history*, which includes the patient's mental attitude to his life and his work. Here it is well to begin with what may be grouped together as the patient's physical and emotional environment, his surroundings, both at home and at work, and his habits. One should try to visualize the life of one's patient, sharing his emotions and viewing step by step his home, his family, his daily habits, his diet, and his work. It will often be found advantageous to ask the patient to give a brief account of a typical day.

Inquire into (a) the exact nature of his occupation (not merely the name of his trade but what precisely his work involves) and whether or not it exposes him to injurious influences; former occupations should also be noted. Sometimes one should inquire into a patient's business affairs, his ambitions, anxieties, quarrels, and, in general, his attitude to his work. (b) His home surroundings, their sanitary conditions, and the possible existence of overcrowding. What pets does he keep? Where did they come from and were they recently imported? (c) His domestic relationships, his feelings about the other members of his family, his own psychological make-up, his interests, his hobbies, his hopes, his fears, the holidays he gets and whether he enjoys them, the amount of exercise he takes, the games he plays, and, in general, the sort of life he leads and the sort of person he is. (d) The nature of his food, and the extent of his indulgence in alcohol and tobacco, and any change in his smoking habits over the years. (e) Whether or not he has lived abroad, and if so, in what part of the world and whether he was ill there.

One should next take up the question of the patient's *previous health*. Find out what illnesses he has had, when he had them, and their duration. Be careful not to accept uncritically a diagnosis of a previous illness: find out by whom the diagnosis was made, and confirm this if possible by a few questions as to the symptoms experienced. Find out the name of any hospital where the patient has been treated; the approximate date of the illness; the patient's home

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address at the time; and, in the case of a married woman, her name before marriage. Adults should be asked whether they have had a venereal disease and, if necessary, whether they have run the risk of it. In the case of female patients, questions should be put as delicately as possible. Female patients should also be asked about menstruation. In the majority of cases menstruation occurs every 28 days, but the intervals may be longer or shorter according to the patient's habit. If menstruation has ceased, one must inquire how long it has been absent. Normally the cessation of menstruation, or menopause, should not occur until about the 45th year or later. It is also necessary to inquire whether the patient is losing more or less blood than usual. The menstrual flow is to be regarded as abnormal if it lasts for fewer than two or more than eight days. The presence or absence of pain at the periods must be noted. The patient should also be asked whether she has been taking contraceptive pills.

We are now ready to obtain the history of the *present disorder*. Ask how and when it began, getting dates of the chief events if possible, and whether suddenly or gradually; what was the first thing the patient noticed wrong; what has been the order of appearance of his or her symptoms, and which of them are most troublesome at the present time. The use of medical or pseudo-medical terms should be gently but firmly discouraged. A patient, for instance, who says the complaint is 'catarrh' or 'rheumatism' should be asked to describe what exactly it is that he or she feels wrong. Find out whether or not he or she has already been under treatment, and, if so, what has been done. It is often useful to ask why the patient came to the doctor or the hospital at this particular time. The answer is sometimes surprising.

This completes the general interrogation, and includes the chief facts that have to be inquired into in every case.

### 2 The Special Interrogation

This must be modified according to circumstances. It is only by experience that one can tell what it is essential to ask in each individual case. In order to help the beginner, there follows a scheme of interrogation which he can pursue when he has reason to suppose that the patient's general symptoms point to disorder of any particular system or organ. Such a scheme is necessarily far from complete, and one is not able in such a work as this to explain *why* such and such questions should be put. The reasons for the questions will become apparent in time.

## 1 Alimentary System and Abdomen

(a) Symptoms point to an affection of the *upper alimentary tract*. Inquire about:

**Pain.** What is its severity and exact site? Is it localized or diffuse? Does it radiate in any particular direction? For how long has he had it? Does he have intervals of freedom? If so, for how long? What is its relation to meals (if any)? Does it wake him at night? What things aggravate it? What affords relief (e.g. food, alkaline powders, vomiting)? Distinguish especially between 'pain' and mere 'sense of discomfort' or 'fullness'.

**Appetite.** Is it increased or reduced? If reduced, is his appetite really bad, or is he afraid to eat on account of pain?

**Meals.** Arrangement of these; the nature of the food. Is the diet adequate in amount and in essential constituents? Are meals irregular on account of shift work?

**Weight.** Is the weight increasing, decreasing or remaining stationary? What is the most the patient has ever weighed?

**Vomiting.** Frequency. Its relation to pain; does it relieve pain or not? Distinguish between vomiting (contraction of abdominal muscles and diaphragm) and regurgitation (reverse peristalsis in stomach).

**General characters of vomited matter.** Its amount and colour. Does it ever contain blood? Does it ever look like 'coffee-grounds'; is it ever sour and frothy? Does it contain residues of food taken the day before?

**Flatulence.** Relation to particular articles of food. Does the wind tend to escape downwards or upwards? Does either form relieve the symptoms?

**Water brash.** Does he ever experience excessive secretion of saliva into the mouth, with regurgitation of mouthfuls of clear, tasteless fluid?

**Heartburn.** Does he suffer from pain behind the sternum? Does it come on especially when he is lying down?

**Dysphagia.** Is there any difficulty in swallowing? If so, where does the food appear to 'stick'? Is it worse with liquids or with solids?

(b) Symptoms point to an affection of the *intestines*. Inquire about:

**Diarrhoea.** Number and time of occurrence of motions during the day; their relation to meals or to special articles of food. Colour of the motions; are they formed, unformed, porridge-like, frothy, or frankly watery? Do they float in the lavatory pan or are they difficult to flush away? Has he ever passed any blood or slime? Is there pain during defaecation? Does the patient use purgatives or does he take anything else, e.g. beer, likely to produce loose motions?

**Constipation.** What is his usual habit? Are the bowels opened regularly, and if so, how often? Has there been recent change in bowel habit? How long since the last motion? Does the constipation alternate with diarrhoea? If so, can this be explained by the taking of purgatives? Has he any gripping pain? Has he had any vomiting? Does he take codeine in any form?

*Pain.* Site, radiation, and character? Persistent or intermittent? Where is it felt worst? Is it relieved by defaecation or by the passage of flatus?

(c) Symptoms point to an affection of the *liver* or *gall-bladder*—e.g. patient is jaundiced, or has pain in the region of the liver. Inquire about:

*Pain.* Its site. Has he ever had any attacks of very severe pain, coming on suddenly and lasting for a few hours? If so, did the pain radiate, and in what direction? Was there vomiting with it? Was he yellow after it subsided? Has he ever had pain in the tip of the shoulder?

Has he noticed any change in the colour of the urine or faeces? Does the skin itch?

Inquire also regarding his digestion on the lines of the interrogation already laid down for affections of the stomach.

## 2 Circulatory System

If the symptoms point to an affection of the circulatory system inquire about:

A history of rheumatic fever, chorea, scarlet fever, or diphtheria. (If a child, ask also about sore throats and 'growing pains'.)

The following subjective sensations:

*Dyspnoea.* When does it come on? Is it present at rest or only on exertion? What degree of exertion is necessary to produce it? Does he have attacks of breathlessness at night? Does he have to sit up in bed, or can he sleep lying down? *Precordial pain* or distress; its exact site and character; does it radiate or not? If so, in what direction? What precipitates it, and what, if anything, relieves it? *Palpitation*; what brings it on, and how long does it last? Does the heart give an occasional thump now and then? Has the patient ever felt his own pulse during an attack?

Do the feet swell?

## 3 The Blood

If the symptoms and appearances point to an affection of the blood, inquire about:

Family history of bleeders. Has he had any loss of blood? Has he been taking aspirin in any form? Are the stools ever black? Has he bleeding piles? (If a woman—is menstruation excessive or diminished?) What kind of a diet does he eat? What drugs has he been taking and to what chemical substances is he exposed in his work or home?

Such subjective sensations as breathlessness on exertion; headache; giddiness; palpitation.

Do the feet swell?

#### 4 The Respiratory System

If the symptoms point to an affection of the respiratory system, ask about:

Family history of asthma, hay fever, or other allergies, or chronic bronchitis? Any tuberculosis amongst family or friends? It may even be necessary to inquire about the occupation of other members of the family, who may bring asbestos or other dusts home on their clothes.

The patient's job? Is he exposed at work or at home to any mineral, vegetable, or animal dusts that could cause pulmonary disease? In particular to silica, asbestos, cotton, and mouldy hay? Does he keep cats, horses, or birds as pets? Does he grow any particular flowers? Where does he live, in the foggy town, or in the country where the air is cleaner but may be laden with pollen? Does he smoke? Details of smoking habits past and present. Patients who smoke lightly or not at all at present may have been heavy smokers in the past.

In the past history, details of infantile eczema, skin rashes, or allergy are important, also certain childhood diseases such as measles; or whooping cough, which may be associated with pneumonia or even subsequent bronchiectasis. Any previous episodes of pleurisy or pneumonia? Any nasal catarrh or rhinitis? Diseases of the nose and nasal sinuses are often associated with pulmonary disease.

Finally, certain general symptoms may be associated with pulmonary disease. Inquiries should be made about fever, night sweating, and weight loss, all of which may accompany tuberculosis. Weight loss is also often associated with respiratory failure and carbon-dioxide retention (p. 162).

*Cough.* Whether dry or productive. Worst at which time of day? Worse by any particular conditions, such as cold, dust, or pollen? Painful or not?

*Sputum.* Quantity. Most produced at what time of day? Consistency, colour, and odour. Purulent or not? Ever blood-stained, and if so whether with streaks or clots, and on how many occasions?

*Dyspnoea.* Under what conditions is it present? At rest, or after varying degrees of exertion? Various 'grades' of dyspnoea have been described, but for the student it is best to inquire from the patient what sort of activity (e.g. walking upstairs, running for a bus, etc.) produces dyspnoea. Patients with severe pulmonary disease may be dyspnoeic at rest. They may also be dyspnoeic at night. This is not because lying down alters the amount of blood in the lungs (as with cardiac patients, who are orthopnoeic), but usually for one of two other reasons. Either nasal secretions may trickle down the back of the throat into the bronchi, which they irritate, to produce bronchospasm; or because the sticky bronchial mucus which can be a feature of asthma or bronchitis forms in sufficient amounts (and is not

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**expectorated**) to form a plug blocking a relatively large bronchus, so that air cannot reach certain areas of lung.

**Wheeze.** Wheezing may be associated with dyspnoea. The student should inquire when wheeze occurs. Is it constant or intermittent? Does anything provoke it? Is it worse at any particular time of day or night?

**Chest pain.** Where is it? Is it aggravated by deep breathing or coughing? Was it associated with increase in cough, sputum, or dyspnoea? Was the onset sudden, as is spontaneous pneumothorax?

### **5 The Urinary System**

If the symptoms point to an affection of the *kidneys*—e.g. oedema—or *urinary system*—e.g. pain on micturition, ask about:

History of tonsillitis or previous renal disease. Family history of renal disease or high blood pressure?

Has he any pain in the lumbar region or any attacks of acute pain shooting down into the groin or testicles?

The following remote symptoms:

Headache, vomiting, drowsiness, paralysis or fits, dimness of vision, dyspnoea.

Does the face ever look puffy in the morning? Are the ankles swollen? What is the state of the bowels?

Inquire regarding micturition, as follows:

**Urine.** Is it altered in amount? Does he have to rise in the night to pass it?

Is it altered in colour? Is it clear or turbid when passed? Ever any blood in it? If so, at what period of micturition is it present? Is it frothy?

Is there any increased frequency of micturition? Is the increase by day or by night? Is there an increase in volume passed? Is frequency associated with undue thirst?

Is there any pain during micturition? Is it before, during or after the act? What is its character, and where is it felt?

### **6 Skin Diseases**

Inquire carefully into the patient's personal habits as regards diet, clothing and washing. What is his occupation? Does he handle chemical substances or other irritants? Ask if he has been taking any drugs recently. It may be necessary to inquire carefully regarding syphilis. Does the eruption itch? If so, when is the itching worst? Did the eruption appear all at once or in crops? Does he suffer from asthma, hay fever, or any other allergic conditions?

### **7 The Nervous System**

If symptoms point to an affection of the nervous system, ask about:

A *family* history of mental illness, paralysis, or fits.

The nature of the patient's work; is he exposed to any poisons—e.g.

lead, mercury, manganese, carbon disulphide, or other volatile substances? Syphilis and alcohol should be inquired about with special care. Has he been exposed to tropical infestations?

In cerebral cases it is important to inquire regarding discharge from the ear, and the possibility of a head injury.

Should he complain of *fits*, the following questions should be asked:

- Age at first fit? Describe the first fit. When did the second occur? What has been shortest and longest interval between the fits? Do they occur in sleep or not? Has he any premonition or aura? What is its character? Does the patient go rigid? Does he lose consciousness? Is the onset sudden or gradual? Are convulsions present? Are they general or local? Where do they begin and end? Does he fall? Has he ever hurt himself? Does he bite his tongue, micturate, or defaecate during the fit? Are there any after-symptoms, such as sleep, headache, automatism, or paralysis? Is there any subsequent mental disturbance? Because these patients are seldom clear as to the exact nature of their fits, it is essential to interview separately a reliable person who has seen the patient in a fit. The word 'fit' is undesirable at a first questioning and 'attack' is preferable.

If he complains of *paralysis*, inquire regarding:

Symptoms of heart disease, hypertension, or diabetes (*see Circulatory and Urinary Systems*). Had he any premonitory symptoms before the onset? How did the paralysis come on? Suddenly or gradually? Has he any headache or vomiting? Where is the headache situated? (The method of eliciting other subjective symptoms of nervous disease is considered along with the investigation of the cranial nerves in Chap. 9, p. 224.)

## 8 The Mental State

The following special scheme for cases presenting mental symptoms will often be found useful:

The mental condition of the patient is assessed by (1) what we observe of his behaviour; (2) b, the way he responds to our questions; (3) by the information which is given us by his relatives and friends. Only points about the behaviour of the patient are mentioned here. Our picture of him rests not only on how he behaves, but on how he tells his own story.

Is he reasonably clean and neat, or unkempt of hair and nails and unshaven?

Is he reacting in the expected way to his complaints, or is he over-talkative or almost mute? Does he display unwarranted hilarity, fussiness, or resentment? Is he placid or excited? Are both his conversation and his movement slower or quicker than one would expect? Does he know where he is? What is the date and how old he is? And who he is? (Is he orientated, that is, in space, time and person?) Is

his consciousness clouded (confusion)? Is he conscious, but does not answer questions (stupor)? Or is he unconscious (coma)? Is he incontinent of faeces or urine?

Is he idle or active, co-operative or not? Can his attention be held or does it wander? Does he answer questions directly, or slowly, or make unexpected replies?

Does it appear that he is intelligent or dull-witted? Is the story which he tells to the point, or discursive and vague, and is this due to some temporary clouding, or to permanently poor mentality?

Any other information suggesting a disturbed mental state should be included in the clinical note. The detailed way in which a fuller psychiatric examination is to be conducted will be found on p. 216.

## 9 The Skeletal System

The symptoms point to an affection of the bones or joints:

Inquire for previous manifestations of rheumatism, rheumatic fever, or gout and for the presence of urethral discharge in a male or leucorrhœa in a female. Tuberculosis and syphilis may occasionally be important. Is there a family history of gout? Has the patient been exposed to rubella?

If there is pain referred to a bone, ask whether it is worse during the day or during the night. If the pain is in a joint, ask whether it is present constantly or only when the joint is moved. Does the pain shift from one joint to another?

## 10 Occupational History

The following notes on taking of an occupational history will be found helpful.

In clinical practice the occupational history is often valuable, and there are few surer and quicker means of gaining a patient's confidence than the display of an intelligent knowledge of his job. It is a wise rule to take the occupational history from the time the patient left school. Record the dates and items of all subsequent jobs. He may be exposed to a noxious substance responsible for his ill-health in his present occupation, but this should not be assumed. A man describing himself as an ice-cream vendor may have cancer of the skin of the hand due to work in the pitch-beds of a gasworks 20 years before, or a mesothelioma due to exposure to asbestos many years before. Ask the patient the name of his trade, the processes employed, the tools used, and the substances handled. The name of an occupation may be misleading, for different names are used for the same process in different places.

It often happens that workmen and foremen refer to chemical substances by their popular names and not by their chemical names. Examples of such names are lunar caustic for silver nitrate, chtome yellow for lead chromate, wood spirit for methyl alcohol, and oil of mirbane for nitro-

benzene. The man may be ignorant of the nature of a substance he uses, and know it only by a trade name. In such cases it is best to communicate with his works manager and ask what is the nature of the substance in question.

Question him as to the general conditions at his place of work. If necessary ask him to sketch on paper a plan of his workshop and of the apparatus he uses. Is the job dusty, and if so what tools make the dust? Are there fumes or vapours, and if so what are the chemical substances involved? Most of the toxic substances encountered in the dangerous trades enter the body by inhalation. Ask whether a hood is installed over his bench, and whether it is connected to a suction system. Ask about the provision of protective clothing at his place of work. Does he wear a special suit, gloves or goggles, and why? Finally ask whether any similar illness has befallen a fellow workman.

Whenever serious doubts and difficulties arise, it is advisable to visit the factory in order to ascertain the conditions of work on the spot. In difficult cases the practitioner should enlist the help and advice of HM Factory Inspectorate, through the Senior Medical Inspector, Baynards House, Chepstow Place, W2 (01—229 3456).

Other aspects of the history are no less important. A particular illness may render a man temporarily or permanently unfit to do his work. The doctor should know that conditions peculiar to certain trades may cause disease which predisposes to infection. Thus, silicosis leads to an excessive mortality from pulmonary tuberculosis and also from pneumonia. Diseases other than infections may be involved. For example, a heavy mortality from cirrhosis of the liver as well as from tuberculosis exists among publicans, barmen, brewers' draymen, and others who have ready access to alcohol.

The doctor should have regard for his patient's work, even when he is suffering from a disease which is non-occupational; one must know whether a man does a job which makes him a danger to others. A dairyman with open tuberculosis can contaminate milk with tubercle bacilli by coughing into it, and those who handle food can initiate outbreaks of typhoid fever, dysentery and cysticercosis by acting as carriers.

## 11 Children

If the patient is a young child, the following special questions should be put to the mother or other responsible person:

How many other children are there in the family? What are their sexes and ages? Have there been any miscarriages or stillbirths? If so, when? Is there a history of illness in the parents or siblings, or in the parents' near relatives?

Was the mother well during pregnancy, and did she take any drugs? Was this a full-term infant? What was its birth weight? Was it born at

home or in hospital, and was the labour normal? Were there any unusual symptoms, such as jaundice or cyanosis, in the newborn period?

Was the baby breast-fed, and for how long? If bottle-fed, what type of milk was used? Were vitamin supplements given? When was mixed feeding introduced? Was there a satisfactory weight-gain in infancy? What immunizations were given, and when?

It is particularly important to inquire about the 'milestones of development' (p. 346). When did the baby first smile, sit up, walk, and talk? When did he acquire control of bowels and bladder?

What are the child's present habits with regard to eating, sleeping, bowels, and micturition? What is his general behaviour like in comparison with other children of the same age? If the child is of school age, does he attend school regularly, does he get on well with his lessons, and does he like school?

Has the child ever been separated from his mother? If so, when and for how long? What is the social background? Are the living conditions satisfactory? Does the mother go out to work? Is this an immigrant family? If so, where do they come from, and how long have they been in this country?

Finally, inquire about previous illnesses, their nature and severity, and the ages at which they occurred—*infectious diseases, fits, bowel disturbances, upper respiratory infections, discharging ears*. If there is a history of cough, was it spasmodic, associated with vomiting, particularly bad at night? And was there a whoop? What drugs has the child received? Has the child ever been in hospital?

## II THE PHYSICAL EXAMINATION

First investigate the patient's general state, as in Chap. 2. Then go on to an examination geographically, from above downwards, afterwards writing down the findings under the various systems. The results yielded by inspection, palpation, percussion, and auscultation should always be stated in that order.

One more point regarding case-taking remains to be emphasized, and that is to note negative as well as positive facts. It is often quite as essential, for example, to state that such a symptom as dyspnoea is absent as to record the fact of its presence.

It need hardly be said that the examination should be carried out as gently as possible, all unnecessary exposure, exhaustion, or chilling of the patient being carefully avoided. If the patient is suffering from severe or acute disease, it may be advisable to postpone all physical examination other than that which is absolutely necessary for the

diagnosis of his condition, or for guidance in treatment. Severely ill patients must not be put to any unnecessary exertion or disturbance.

### Case-Taking Scheme

#### 1 Interrogation

Name. Age. Occupation. Married or single. Address. Date of coming under observation.

#### Presenting complaint

#### Duration

**Family history.** Inquire regarding parents, brothers and sisters, and patient's own children. Note state of their health; or the cause of their death, with age at which they died.

•  
**Personal history. Social history.** Environment, nature of work and its surroundings. Hygienic conditions at home. Mental attitude, interests, hobbies, fears, and ambitions.

*Previous illnesses or accidents* (if any), with their time of occurrence, duration, and results.

*Habits*, as to exercise, food, alcohol, and tobacco.

*Present illness.* Time and mode of its origin, the order in which symptoms appeared, and the chief symptoms which trouble patient now; treatment (if any) already employed.

#### II Physical Examination

**General.** While taking the history and in the course of the examination the following points should be observed:

General aspect of the patient—appearance of 'illness'.  
intelligence.  
co-operation.  
expression.  
position.  
build.

Temperature and pulse rate.

Skin colour; cyanosis; anaemia, jaundice; pigmentation.

Skin eruptions.

Oedema; wasting; obesity.

Body hair.

Deformities; swellings.

The main examination begins with the head and neck and proceeds down the body.

*The Head and Neck*

Skull: shape; percussion and auscultation.

Hair.

Eyes: Exophthalmos; ptosis; oedema of lids.

Conjunctivae: anaemia; jaundice; inflammation.

Pupils: size; equality; regularity; reaction to light and accommodation.

Eye movements: strabismus; nystagmus.

Fundi.

Acuity of vision and visual fields.

Ears: Inspection of drums and simple tests of hearing.

Face: Function of motor and sensory parts of fifth nerve.

Function of seventh nerve.

Mouth and pharynx (a tongue depressor and torch should be used):

Breath.

Lips: colour and eruptions.

Tongue: protrusion and appearance.

Teeth and gums.

Buccal mucous membrane: colour and pigmentation.

Pharynx: movement of soft palate; state of tonsils.

Neck: Lymphatic glands.

Venous distension and palpation of carotid vessels.

Thyroid.

Movements and muscle power.

*Upper Limbs*

General examination of arms and hands.

Fingernails: clubbing; koilonychia.

Pulse: rate; rhythm; volume; character; thickness of arterial wall of radials and brachials.

Axillae: lymphatic glands.

Blood-pressure.

Nutrition; power; muscle tone; tendon reflexes; co-ordination and sensation.

Joints.

*Thorax*1. *Anteriorly and laterally*

Note the type of chest and any asymmetry.

Rate, depth, and character of respiration.

Pulsations.

Dilated vessels.

Define the position of the trachea by palpation.

Look for the cardiac impulse.

Palpate cardiac impulse.

**Palpate** over praecordium for thrills, and over any visible pulsations.  
Define the area of cardiac dullness by percussion.

**Auscultate** the heart sounds.

**Palpate** respiratory movements.

**Estimate** tactile vocal fremitus.

**Percuss** the lungs.

**Auscultate** the breath sounds.

**Estimate** vocal resonance.

## 2. Posteriorly (patient sitting)

**Inspect** and **palpate** chest expansion—apices and bases.

**Estimate** vocal fremitus.

**Percussion** of chest.

**Auscultation**: breath sounds; vocal resonance.

**Note** and **palpate**: deformities of spine; spinal movements and the presence or absence of tenderness; oedema of lumbar pads.

**Palpate** the neck from behind: cervical glands; thyroid.

## Abdomen

**Inspection**: size; shape; distension; symmetry; movement of abdominal wall; dilated vessels; scars; umbilicus, visible peristalsis; pulsations; pubic hair; hernial orifices.

**Palpation**: tenderness; rigidity; splashing; increased resistance to palpation; masses; fluid thrill; liver; spleen; kidneys; abnormal masses.

**Percussion**: when necessary.

**Auscultation**: when necessary.

Hernial orifices: patency; impulse on coughing.

Inguinal glands.

Genitalia: penis; scrotum; spermatic cord and testicles.

Abdominal reflexes.

Rectal examination.

Vaginal examination, if indicated.

## Lower Limbs

General examination of legs and feet.

Oedema—pitting of skin over ankles and thighs.

Nutrition; power; muscle tone; tendon reflexes; co-ordination and sensation.

Joints. Pulsation in dorsalis pedis and posterior tibial arteries.

## Examination of Excreta

1. Urine

2. Sputum

3. Stools naked-eye description, including amount.

4. Vomitus

In addition to the above scheme, a full neurological and/or psychological examination may be necessary. Neurological examination is described in Chap. 9, and a brief outline of psychological examination is included in this chapter.

**Writing out the physical examination.** The date must be given. Whatever the order in which the examination was performed, it should be described systematically. Relevant negative findings are as important as positive ones.

*General.* Begin with a description, in one or two sentences, of the general appearance and mentality.

Record the temperature, pulse and respiration rates.

Record the state of:

Mucous membranes.

Skin.

Mouth.

Nails.

Hair.

Thyroid gland.

Lymphatic glands.

Bones and joints.

Note the presence or absence of anaemia, cyanosis, jaundice, oedema, clubbing of the fingers, and the condition of tongue, fauces, tonsils, and teeth.

#### *Cardiovascular System*

Pulse.

Condition of other arteries.

Neck veins.

Pulsations and their character.

Heart: position of cardiac impulse and its character; presence or absence of thrills; character and intensity of heart sounds; and their propagation.

Blood-pressure.

#### *Respiratory System*

Rate and character of respiration.

Position of trachea.

Shape of chest; symmetry.

Results of inspection, palpation, percussion, and auscultation, noting areas affected, if any abnormality is found.

Sputum: quantity and character.

*Abdomen*

Inspection: shape; distension (general or local); movements with respirations; distended veins; umbilicus; pulsation; visible peristalsis.

Palpation and percussion: tenderness; rigidity; liver; spleen; kidneys; any other abdominal masses; ascites; hernial orifices.

Auscultation: peristaltic sounds; arterial bruits.

Spine.

In men: genitalia: rectal examination.

In women: vaginal and rectal examination as indicated.

*Nervous System*

General. Mental state; intellectual functions; emotional state; speech and articulation; gait.

Cranial nerves. In any neurological case a full description of the examination of the cranial nerves, taken in their order, should be given. Bruits over the neck or skull should be noted.

Upper limbs.

Trunk.

Lower limbs.

Physical signs should be described in the following order:  
wasting and fasciculation; power; tone; co-ordination; reflexes;  
sensation.

*Urine*

Colour; reaction; specific gravity, sugar; protein; blood; deposit (microscopic examination).

*Stools, Sputum, and Vomit* should be described, where necessary.

**Results of special investigations****Diagnosis****Prognosis***Notes of Treatment and Progress*

(Daily notes in acute cases; in others make a note of progress every three days.)

State on discharge, with note of drugs being taken.

If patient died, add notes of post-mortem (if held).

## **2** GENERAL CONDITION AND APPEARANCES

It should hardly be necessary to say that a proper physical examination demands the absence of noise and an adequately warmed room. Good daylight is essential for the perception of colour changes such as those present in anaemia and cyanosis, while slight degrees of jaundice cannot be seen at all in artificial light. For a complete physical examination, the patient should be stripped completely and covered with a blanket or dressing-gown, though only the part or parts actually being examined need be uncovered at any one time. In practice, patients are often examined incompletely stripped, i.e. wearing pants or trousers, but this can lead to mistakes. If the examination is attempted in a cold room, the patient usually shivers. This is distressing to him, and makes most forms of examination useless. In particular, it is impossible to auscultate the chest, as strange noises are produced in the stethoscope by shivering muscle. Ideally another woman should be present when a male doctor is making any examination of a female patient, and this is essential in the case of rectal and vaginal examinations, both to reassure the patient and to protect the doctor from subsequent accusations of impropriety.

Physical examination proceeds by the time-honoured methods of inspection, palpation, percussion and auscultation, applied with appropriate modifications to the different systems and parts of the body. It may be said, however, that the experienced doctor begins his examination as soon as the patient enters the room, just as he continues taking a history till the patient leaves it. Examination of systems may provide information about the state of organs and functions, but it is also important to try to view the patient as a whole.

Thus, from the moment of meeting the patient, and while taking the history, the experienced doctor will be making observations. The patient's gait as he walks into the room, his posture when sitting or standing, his dress, speech, manner of answering questions, level of intelligence and emotional state may all give valuable information. What kind of a person is this? What is his background? What is he

thinking? What is he feeling? Is he in general a happy person or an unhappy one? He is probably anxious. Is this anxiety proportionate to the sign of disease present and reasonable, or disproportionate and unreasonable? What is the real reason for his coming to see a doctor at this particular time? Doctors vary in their ability to answer questions of this kind, and the skill to do so can in any case only come with actual practice. Students should realize that an important function of their periods of clerking in wards and out-patient departments is to give them an early opportunity to acquire something of this ability, which cannot be gained from any textbook or lecture.

One of the first things to observe about a patient in bed is his *attitude*. In health a person lies in any manner in which he feels comfortable. He changes his position without difficulty from time to time, and has no hesitation in moving if he slips from his pillows or feels otherwise uncomfortable. In disease this ability to maintain a comfortable position is lost in various degrees; and severely ill patients become helpless, so that they slip down passively into positions which may be very uncomfortable.

Patients with heart failure are often unable to lie in a horizontal position, and must sit more or less upright in a chair or propped up with pillows. This difficulty in breathing when lying flat is referred to as *orthopnoea*.

In *abdominal disease* the aspect of the patient is often characteristic. When *peritonitis* is present he lies on his back, breathing shallowly to avoid the pain that movement produces. Sometimes one or both legs are drawn up.

In *colic* there is often great restlessness, which contrasts vividly with the fixed attitude of peritonitis; in *renal colic* the patient rolls about and tries one position after another in a futile search for relief.

Patients who are attacked by acute rheumatism or other painful joint diseases have a peculiar aspect of helplessness, the limbs lying motionless, the joints being swollen, stiff, and painful.

Various *diseases of the nervous system* produce characteristic attitudes; peculiarly important is that of meningitis, where in the severest cases the neck is bent backwards so that the head seems to bore into the pillow.

When possible, the physician should not only study his patient in bed, but should also see him up and walking. The forward stoop and festinant gait of *paralysis agitans* are as characteristic as the mask-like *facies*, tremor, and *pill-rolling movements*. The trunk is thrown back

## 20 GENERAL CONDITION AND APPEARANCES

in hypertrophic muscular dystrophy, in pregnancy and in massive abdominal tumour, and often bent forward when abdominal pain is present.

When the patient walks, any peculiarity in his *gait* must be observed. The more important types of gait are described in Chap. 11, but it should be remembered that alterations may be due not only to diseases of the muscular and nervous systems; a painful toe or local condition in any of the joints of the lower limbs may produce characteristic effects.

Notice should be taken of the *dress*. Apart from insanity, where the patient's clothing may be dishevelled or grotesque, one may discover indications of a local or general change in his bulk, or his shoes may wear unevenly in consequence of some abnormality of gait.

The *general development* and *nutrition* of the patient should be compared with the examiner's opinion of the normal for the patient's age, sex, and physical type. In medical examinations for insurance purposes it is usual to record the chest measurement during inspiration and expiration at the level of the nipples (in male subjects), and also the waist measurement. If the latter exceeds the former, either the subject is unduly fat or the abdomen is enlarged by disease, usually by the presence of fluid (ascites). Wide variations in build are compatible with good health. The tables on page 21 show ideal weights for different heights and builds, as used for insurance purposes.

The state of nutrition may be assessed by the use of these tables or by simple observation. In health there is a fair quantity of subcutaneous fat, the muscles are of moderate size and firm texture. The skin is elastic and neither very moist nor very dry. In obesity there is an excess of subcutaneous fat, especially over the abdomen and thighs. In wasted and emaciated patients there is a loss of subcutaneous fat and the skin is loose.

The *expression* of the patient may yield information of importance and sometimes indicates his real feelings better than do his words. In exophthalmos the *eyes* are prominent, often showing a ring of sclerotic above the cornea; or the prominence may be due to a high degree of myopia. In wasting disease or in profound collapse, such as is found in any state of severe dehydration, the eyes are sunken.

The *lower eyelids* are puffy and oedematous, especially in the morning, when the patient is suffering from acute nephritis or the nephrotic syndrome; and a similar appearance is sometimes noted in patients who are suffering from severe paroxysms of cough. It is characteristically

**GENERAL DEVELOPMENT**  
**IDEAL WEIGHTS FOR MEN (AGES 25 AND OVER)**

21

ft. in.	<i>Height (with shoes)</i>	<i>Weight in Pounds (as ordinarily dressed)</i>		
		<i>Small Frame</i>	<i>Medium Frame</i>	<i>Large Frame</i>
5 2.	.	116-125	124-133	131-141
5 3.	.	119-128	127-136	133-144
5 4.	.	122-136	130-140	137-149
5 5.	.	126-136	134-144	141-153
5 6.	.	129-139	137-147	145-157
5 7.	.	133-143	141-151	149-162
5 8.	.	136-147	145-156	153-166
5 9.	.	140-151	149-160	157-170
5 10.	.	144-155	153-164	161-175
5 11.	.	148-159	157-168	165-180
6 0.	.	152-164	161-173	169-185
6 1.	.	157-169	166-178	174-190
6 2.	.	163-175	171-184	179-196
6 3.	.	168-180	176-189	184-202

**IDEAL WEIGHTS FOR WOMEN (AGES 25 AND OVER)**

ft. in.	<i>Height (with shoes)</i>	<i>Weight in Pounds (as ordinarily dressed)</i>		
		<i>Small Frame</i>	<i>Medium Frame</i>	<i>Large Frame</i>
4 11.	.	104-111	110-118	117-127
5 0.	.	105-113	112-120	119-129
5 1.	.	107-115	114-122	121-131
5 2.	.	110-118	117-125	124-135
5 3.	.	113-121	120-128	127-135
5 4.	.	116-125	124-132	131-142
5 5.	.	119-128	127-135	133-145
5 6.	.	123-132	130-140	138-150
5 7.	.	126-136	134-144	142-154
5 8.	.	129-139	137-147	145-158
5 9.	.	133-143	141-151	149-158
5 10.	.	136-147	145-155	152-166
5 11.	.	139-150	148-158	155-169

(Metropolitan Life Insurance Company, Statistical Bureau, 1943.)

It will be noticed that tables of this kind make no allowance for so-called 'middle-aged spread'.

## 22 GENERAL CONDITION AND APPEARANCES

present in adults with myxoedema and in children affected with whooping-cough.

The *nose* has a sunken bridge in congenital syphilis. Some alcoholics have red noses, but by no means all persons with red noses are alcoholics. Undue mobility of the alae nasi may be due to nervousness, or may indicate obstruction to inspiration, and is in this respect very important to look for in infants.

The *lips* are pale in anaemia, livid and blue in congestive heart failure. The vesicles of herpes febrilis on the lip are very often associated with inflammation of the respiratory tract, and their presence should lead to a search for pneumonia.

The *ears* may show tophi in persons with gout, and may be abnormal in congenital renal disease and deformed in boxers.

The *cheeks* give some information regarding the patient's health. In anaemia and aortic regurgitation they are pale; in some cases of mitral stenosis there is a bright circumscribed flush over the malar bones; in many persons who lead an open-air life they are red and high-coloured; in congestive heart-failure they are also high-coloured, but the colour is of a bluish tint which cannot be mistaken for the red cheeks of weather-beaten people. In some cases of disseminated lupus erythematosus there is a red raised eruption on the bridge of the nose extending on to the cheeks in a 'bat's-wing' distribution.

The *form of the cranium* is considered in Chap. 11.

In addition to the appearance of individual features, the *general expression* of the patient must be noted. Is it animated or apathetic, or has it the absolute vacancy of unconsciousness? Are there wrinkles on the face, or is it smooth; or is one side smooth and the other wrinkled, as one sees it in unilateral paralysis of the seventh nerve? Is the mouth drawn over to one side, and is there any other lack of symmetry between the two halves? The expression may be characteristic of pain, or may show a placidity gainsaying assertions of severe agony. Twitching of the face may be due to a nervous habit or tic, or may be a manifestation of chorea. Some characteristic facies are shown in Plates I and II.

The *colour of the skin* should be noted. The most important abnormalities are pallor, yellowness, pigmentation, and cyanosis. *Pallor* depends on the thickness and quality of the skin, and the amount and quality of blood in the capillaries. It is thus seen in persons with thick or opaque skins who are always pale; in states where the blood-flow in the capillaries is diminished, such as shock, syncope, or left heart failure; and locally in a limb deprived of its

blood supply; or in the fingers or toes when arterial spasm occurs on exposure to cold, as in Raynaud's disease. Generalized pallor may also occur in severe anaemia. Anaemia, however, is to be judged by the colour of the blood rather than that of the patient, and the colour of the skin may be most misleading. That of the mucous membranes of the mouth and conjunctivae gives a better indication, and so does the colour of the creases in the palm of the hand. *Yellowness* may be due to haemolytic jaundice, when the tint is pale lemon-yellow; or to obstructive jaundice, when it may be of a dark yellow or orange tint. In obstructive jaundice there may be scratch marks that result from the itching which the bile salts evoke. *Pigmentation* is found in Addison's disease, where it affects both the skin and the buccal mucosa. Other forms of pigmentation are described in Chap. 8. *Cyanosis* is a bluish colour of the skin and mucous membranes due to an increase in the amount of reduced haemoglobin in the blood. It may be divided into central and peripheral. Central cyanosis results from imperfect oxygenation of blood, as in heart failure and some lung diseases, or from the admixture of arterial and venous blood in the presence of right-to-left or venous-arterial shunts in the heart. In this case the cyanosis is general and the cyanosed extremities are warm. It characteristically affects the tongue. Peripheral cyanosis is due to excessive reduction of oxyhaemoglobin in the capillaries, when the flow of blood is slowed. This may happen on exposure to cold, when there is venous obstruction, or in heart failure. The cyanosed extremity or extremities are then cold and the tongue is unaffected. One should note, however, that the cyanosis of heart failure is often of a mixed type, due to both central and peripheral causes. A similar bluish or leaden colour may be produced by methaemoglobinaemia or sulphaemoglobinaemia, usually due to the taking of drugs such as phenacetin. This should be considered in any patient who is cyanosed but not breathless. Carbon-monoxide poisoning produces a generalized cherry-red discolouration.

It is also important to look for *cutaneous eruptions*, some of which—measles and syphilitic rashes, for example—frequently appear first about the roots of the hair, whilst others have equally distinctive situations. Ulcers and scars should be looked for. Spider naevi and palmar erythema are important in liver disease. The colour and nutrition of the hair, and the dryness or moisture of the skin must be noted. The skin is dry in myxoedema. The perspiring hands of a rheumatic subject are very characteristic.

When an excess of fluid is present in the subcutaneous tissue the condition is known as *oedema*. Thus in acute nephritis an early

## 24 GENERAL CONDITION AND APPEARANCES

symptom is oedema of the face, which is most marked when the patient rises in the morning. In dependent oedema, however, which is typically present in congestive heart failure, and in conditions associated with a low plasma-protein level, the swelling first appears at the ankles and over the dorsum of the foot, and only gradually mounts to the legs, thighs, and trunk. In local venous obstruction, the oedema is confined to the parts from which the return of blood is impeded. In this way one finds oedema of an arm when malignant axillary glands constrict the axillary vein or oedema of a leg in thrombosis of the popliteal or femoral vein. Oedema of the whole upper part of the body may result from intrathoracic tumours. Oedema may be recognized by the pallid and glossy appearance of the skin over the swollen part, by its doughy feel, and by the fact that it pits on finger pressure. In recumbent patients oedema often appears first over the sacrum. In eliciting pitting it is important to press firmly and for a sustained period, or the oedema may be missed.

*Localized oedema* may be due to local changes in capillary permeability, as in angio-neurotic oedema and giant urticaria.

*Subcutaneous emphysema* is uncommon, but if present can be readily recognized by the crackling sensation which is detected on pinching the part affected.

The hands of the patient merit careful observation (Plate V). Notice the strength of grip as he shakes hands; this often indicates improvement or deterioration with considerable accuracy. Their general shape should be noted, along with the state of the joints, the character of the nails, and the presence or absence of finger-clubbing. In osteo-arthritis the finger-joints are often implicated, and bony nodules, known as *Heberden's nodes*, are formed at the basis of the terminal phalanges. In rheumatoid arthritis there is characteristically a spindle-shaped swelling of the interphalangeal joints and later an ulnar deviation of the fingers. *Trophic changes* in the skin may be present in neurological diseases and in disorders of the peripheral circulation (e.g. Raynaud's disease). Characteristic movements or attitudes of the hand may also be seen in athetosis, tetany, and lead palsy. *Tremor* of the hands may occasionally be congenital. In other cases it is due to nervousness, senility, Parkinsonism, thyrotoxicosis, alcoholism, disseminated sclerosis, uraemia, hepatic failure or mercurial poisoning. The methods of studying this symptom are detailed at p. 264. In ulnar paralysis the hand becomes deformed by over-extension of the first phalanges, combined with excessive flexion of the rest, so that a claw-like attitude is produced. This is known as the

'main en griffe'. When the muscles of the thenar and hypothenar eminences have undergone atrophy the hand becomes flattened, and thus somewhat simian. In acromegaly the hands are massive, the fingers being spatulate with square tips, the knuckles are enlarged, and the skin thickened. In *clubbing of the fingers* the tissues at the base of the nail are thickened, and the angle between the nail base and the adjacent skin of the finger is obliterated. The nail itself loses its longitudinal ridges and becomes convex from above down as well as from side to side. In extreme cases the terminal segment of the finger is bulbous like the end of a drumstick. The condition may be congenital or acquired. Gross degrees of clubbing are found in association with severe chronic cyanosis, as in congenital heart disease, and in association with chronic suppuration within the chest, as in bronchiectasis and empyema. Lesser degrees may be found in carcinoma of the lung, pulmonary tuberculosis, and chronic abdominal conditions such as steatorrhoea, Crohn's disease and ulcerative colitis. Clubbing is also an important sign of subacute bacterial endocarditis, when it may be associated with Osler's nodes, tender transient swellings about the size of a pea in the pulp of the fingers and toes, and 'splinter' haemorrhages beneath the nails. In *hypertrophic pulmonary osteo-arthropathy* there is, besides clubbing of the fingers, thickening of the periosteum of radius, ulna, tibia and fibula. This gives rise to swelling above the wrist and ankle. In rare cases these joints themselves are swollen. A transverse furrow in the nail is the record of some former interference with its nutrition and, in the absence of a local cause, may indicate some severe illness in the recent past. *Koilonychia* occurs in iron-deficiency anaemia. The nails are soft, thin and brittle. The normal convexity is entirely lost and replaced by hollowing so great that in some cases drops of water can be run into the concavity of the nail without overflowing.

The *neck* should always be inspected and palpated. Swellings in the neck are usually felt best from behind. Note:

1. The state of the *lymphatic glands*. In infected conditions of the tonsils the glands at the angles of the jaw are enlarged, and those below the jaw in cases of malignant disease in the mouth. Glands draining an inflammatory focus are usually tender. Enlarged tuberculous glands may occur in groups or in long chains beside the sternomastoid, and scars may mark the points of past suppuration. In 'Hodgkin's disease' and other <sup>of</sup> *reticuloses* the glands are enlarged and discrete. In lymphatic leukaemia there may be great enlargement of the glands on both sides. In secondary syphilis the glands under the

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upper part of the trapezius are often palpable. If enlarged glands are found either in the neck or elsewhere, it is important to observe whether they are firm and distinct, or fused together, whether fluctuation can be elicited, and whether they are adherent to adjacent structures.

2. *The thyroid gland.* Inspect the neck for any general or local enlargement of the gland, and observe its movement with the larynx as the patient swallows. Patients find this easier if they are given a glass of water. Then stand behind the patient and palpate the gland with one hand on each side of the neck. Determine if any swelling exists, and if so whether it is uniform or nodular, hard or soft. Sometimes such enlargements exercise considerable pressure on the trachea and occasionally extend into the thorax behind the sternum; at other times, particularly if the disease is malignant, the recurrent laryngeal nerves may become implicated. In cases where there is difficulty in determining whether a tumour is connected with the thyroid, it is helpful to remember that the gland and any tumour connected with it moves up and down during deglutition. Minor degrees of enlargement of the thyroid are often better seen than felt.

3. *Pulsations in the vessels* must be recorded. Any arterial pulsation is both seen and felt as a distinct thrust, whereas venous pulsation is seen but is not felt as a thrill, if it is felt at all. In aortic incompetence the carotid arteries are seen to pulsate forcibly. In aortic stenosis a systolic thrill is felt. Women patients with hypertension sometimes show kinking of the right common carotid artery which simulates aneurysm. The jugular veins may be distended and pulsatile in congestive heart failure. In superior mediastinal obstruction due to retrosternal goitre or malignant neoplasm in the mediastinum, non-pulsatile distended veins may be seen over the neck and upper part of the body: cyanosis and oedema may accompany this sign. Distended neck veins may also be seen in large pericardial effusions.

4. *Boils and carbuncles* are very frequently situated on the back of the neck. As they are sometimes present in cases of diabetes, the urine should be tested for sugar.

The character of a patient's *respiration* is often of great service in diagnosis and prognosis. When the respiratory passages are obstructed the normal quiet respiratory sound is replaced by more or less noisy breathing. When the obstruction occurs in the nose, the breathing is sniffing or bubbling in character. When the soft palate is paralysed, a snoring stertorous sound is produced. When the rima glottidis

is obstructed from any cause, such as spasm or paralysis of the vocal cords or oedema of the larynx, stridor results. If a polypus or other tumour lies between the cords, there may either be stridor or simply noisy breathing. The trachea may have its airway narrowed by pressure from the outside, as in cases of tumour and, rarely, of aneurysm, when the breathing becomes growling, or mucus may obstruct the lumen, producing a rattling sound. The 'death-rattle', which occurs when weakness and insensitiveness combine to prevent any effort at expectoration, is a typical example of the condition. Obstruction in the bronchi sometimes gives rise to audible wheezing sounds. A division of dyspnoeic conditions may be made according as the difficulty in respiration is felt during inspiration or expiration. Most cases of obstruction of the large bronchi are characterized by *inspiratory dyspnoea*, whilst obstruction of the small bronchi produces *expiratory difficulty*. Common examples of the latter are the prolonged expiration in bronchitis with emphysema and in asthmatic attacks. The breathing may be characteristic of diseases quite distinct from those of the respiratory system. Examples of this are stertorous breathing of apoplexy, the hissing expiration of uraemia, and the 'air-hunger' of diabetic coma, which affects both inspiration and expiration.

If *cough* is present, note whether the cough consists of independent explosive expirations or is paroxysmal. In *bronchitis* the cough is at first snort and dry, but as the quantity of secretion increases, it becomes more paroxysmal, and continues until the mucus is expectorated. When due to *early tuberculosis*, the cough is frequent, short, and sharp. It is described as *dry* because there is no rattling of mucus associated with it. Later, when the caseous masses are breaking down, secretion is more copious and the cough becomes moist and paroxysmal. A *nervous cough* generally has the character of single, short, dry explosions, repeated at intervals. Local conditions in the *throat* may be the cause of most troublesome and persistent coughing, and the observer should look for pharyngitis or a post-nasal drip when the patient complains of constant hawking.

In *pleurisy* and *pneumonia* (associated as it often is with pleurisy) the cough consists of solitary dry, hacking, expulsive efforts, suppressed as much as possible to prevent unnecessary pain, but repeated frequently. In *laryngitis* and *croup* the cough may be simply noisy, but more often is either husky or stridulous. When the lumen of the trachea is encroached upon by a *mediastinal tumour* or an *aneurysm*, there may be a very resonant, brassy cough, aptly compared to a

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gander's cry. When once heard, this is almost sufficient to clinch the diagnosis without further examination.

In *hysteria* the cough is often loud and barking, and gives the impression of being produced to attract attention. Such a cough is sometimes associated with hysterical aphonia. *Pertussis*, when fully developed, is distinguished by a most characteristic cough. There is first a long-drawn, almost stridulous inspiration, then a series of short, sharp, expiratory coughs, which follow each other with extreme rapidity. The face turns dark and the veins grow prominent, the child clings firmly to any support it can find, so as to give full play to the accessory muscles of respiration, and when at last the fit of coughing ends, it is followed by a long-drawn whooping inspiration. The severity of the paroxysm induces vomiting, occasionally causes evacuation of the bladder and bowel, and may even lead to rectal prolapse.

*Hiccup*, which results from spasmodic contraction of the diaphragm, is a common disorder. It is usually due to trivial causes; it also may occur in affections of the brain stem, the diaphragm or the peritoneum, and in uraemia, when it is usually persistent.

The *voice* should be studied. It may be nasal either through habit or in consequence of obstruction in the upper airways. The presence of aphonia demands an examination of the larynx (p. 306).

*Temperature*: When taking the temperature, the following practical points must be attended to:

1. The thermometer must be accurate and of NPL standard.
2. The thermometer must be kept in position long enough to allow the mercury to reach the body temperature. It is advisable to exceed the period which the instrument professes to require. The ordinary 'half-minute' thermometer should be left in position for one or two minutes. Collapsed, comatose, and elderly patients, should have the rectal temperature taken with a special 'low-reading' thermometer.
3. In conscious adults the temperature is taken in the mouth or in the axilla. In young children the thermometer should be placed in the fold of the groin, and the thigh flexed on the abdomen; or it may be inserted into the rectum. The temperature of the mouth and rectum is generally at least half a degree higher than that of the groin or axilla. When the temperature is taken in the mouth, the patient must breathe through the nose and keep the lips firmly closed during the observation.
4. Before inserting the thermometer, make it an invariable rule to

wash it in antiseptic or in cold water, and see that the mercury is well shaken down. Wash it again before replacing it in its case. The Centigrade scale is in general use although in Great Britain many people are still more familiar with the Fahrenheit scale (for comparison of the two scales see p. 377).

Normal . . . . .	36.6°–37.2°C or 98°–99°F
Subnormal . . . . .	below 36.6°C or below 98°F
Febrile . . . . .	above 37.2°C or above 99°F
Hyperpyrexia . . . . .	41.6°C " " 107°F
Hypothermia . . . . .	below 35°C or below 95°F

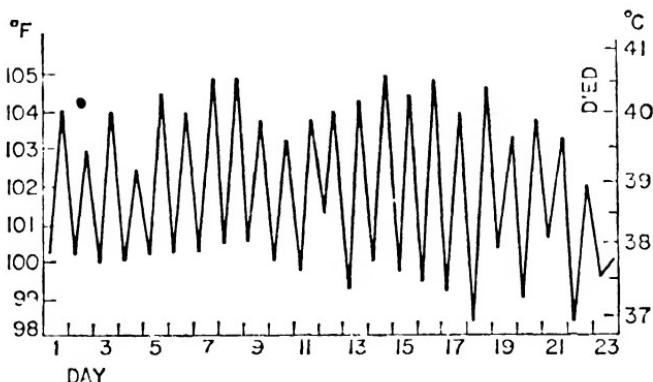


Fig. 1. Remittent fever. Case of pulmonary tuberculosis.

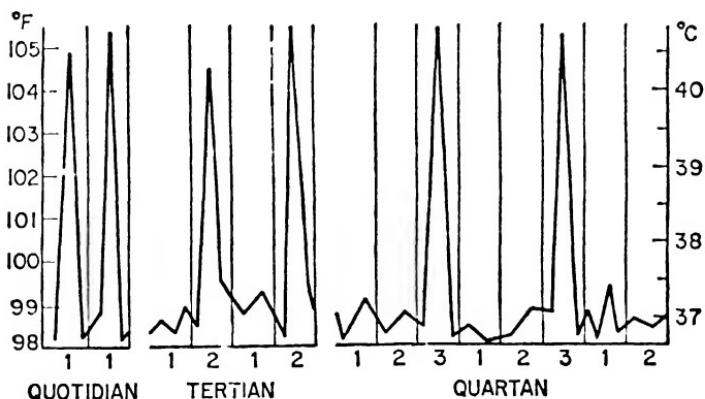


Fig. 2. Intermittent fevers.

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In many conditions, notably acute fevers, there is a disturbance of heat regulation, which may be looked on as the setting of the 'thermostatic' mechanism controlling heat gain and loss at a higher level than normal. While the temperature is rising to this new level, heat is being conserved, the skin vessels are constricted so that the body surface feels cold, and the patient may even shiver violently. This shivering is referred to as a *rigor*. When the higher temperature is reached, heat loss again becomes apparent; the skin vessels dilate and the body surface feels warm. This is the state of affairs present in sustained fever or *pyrexia*.

There are three principal *types of fever*—the continued, the remittent, and the intermittent. When fever does not fluctuate more than about 1°C (or a degree and a half Fahrenheit) during the twenty-four hours, but at no time touches the normal, it is described as *continued*. When the daily fluctuations exceed two degrees, it is known as *remittent* (Fig. 1); and when fever is present only for several hours during the day it is called *intermittent*. In remittent fever the evening temperature is usually higher than the morning one, but in some cases, not infrequently in pulmonary tuberculosis, this type is *inverted*, and the 'remission' occurs in the evening, whilst there is a morning 'exacerbation'. When a paroxysm of intermittent fever occurs daily, the type is '*quotidian*'; when on alternate days, '*tertian*'; when two days intervene between consecutive attacks, '*quartan*' (Fig. 2).

## **3 THE ALIMENTARY SYSTEM AND ABDOMEN**

### **I THE MOUTH, THROAT AND OESOPHAGUS**

#### **The Mouth and Throat**

##### **The Mouth**

For the examination of the mouth the patient should be placed facing a good light. If artificial light is used, it should be thrown into the mouth by means of a head mirror or a torch.

##### **The Teeth**

The *primary teeth* are usually cut in the following order:

The two lower central incisors, sixth to eighth month.

The four upper incisors, eighth to tenth month.

The lower lateral incisors and all the front molars, twelfth to fourteenth month.

The canines (upper first), eighteenth to twentieth month.

Posterior molars, at two to two and a half years

The time of eruption is however variable and tends to be earlier in girls than in boys.

The *permanent teeth* usually appear as follows:

First molars at six years.

Central incisors at eight years.

Lateral incisors at eight years.

Bicuspid (anterior) at nine years.

Bicuspid (posterior) at ten years.

Canines at eleven to twelve years.

Second molars at twelve to thirteen years.

Third molars at seventeen to twenty-five years.

There is again much variation.

The following table shows the numbers of the permanent and the temporary teeth:

## THE ALIMENTARY SYSTEM

PRIMARY	e	d	c	b	a	a	b	c	d	e	20						
	e	d	c	b	a	a	b	c	d	e							
PERMANENT	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8	32
	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8	

Observe the presence of any irregularity or defect or carious disease in the teeth. The absence of a sufficient number of opposing and efficient molars may lead to improper mastication, and so to digestive disorders. Notice whether there is any exposure of the roots, or whether they are surrounded with tartar. The presence of 'Hutchinson's teeth' affords evidence of congenital syphilis. In this condition the two central upper *permanent* incisors are rounded in section and notched at their biting edge. They may also be broader nearer the gum than at the crown, so as to be peg-shaped. They are usually discoloured as well. In the same condition the first permanent molars may be dome-shaped. The notching of 'Hutchinson's teeth' must not be confused with that caused by the habit of holding coat hangers and hair clips between the incisors. This is now much the commonest cause of notching. Enlargement of the lower jaw in acromegaly leads to alteration of the bite so that the lower teeth close outside the upper ones. In endemic fluorosis chalk-white patches appear on the teeth, or they may present a dull unglazed appearance, sometimes with pitting and brown staining.

### The Gums

In *chronic marginal gingivitis*, the gums are retracted, frequently bleed easily and lose their characteristic stippling. *Acute herpetic gingivostomatitis* occurs most commonly in infants and children. Many small vesicles appear on the gums and on the cheeks, palate, tongue and lips. The vesicles rupture to produce shallow ulcers with a yellowish floor and a bright red margin. *Vincent's gingivitis* characteristically destroys the interdental papillae. A thick felted greenish-grey slough is formed, and there is a characteristic odour. In patients exposed to lead compounds, a blue line can often be observed running along the edge of the gum, especially opposite those teeth showing pyorrhoea. This line must be distinguished from similar discolouration due to a black layer of tartar on the teeth. If a wedge-shaped slip of white paper is inserted between the gum and the tooth, the stippled

line of lead poisoning will be rendered more distinct, whereas discolouration due to tartar on the teeth will disappear. A similar but more diffuse line used to be seen after a course of intramuscular bismuth preparations. The gums may be swollen and spongy in scurvy. Hypertrophy of the gums may occur in pregnancy, and in epileptics, treated for long periods with sodium phenytoin (Epanutin). Haemorrhages may be observed in the buccal mucous membrane in thrombocytopenic purpura and acute leukaemia.

### The Tongue

Ask the patient to protrude it. Slight deviation from the mid-line is not uncommon and may be due to asymmetry of the jaws. In hemiplegia deviation towards the paralysed side may be found, while in lesions of the hypoglossal nerve or its nucleus there may be fibrillation of the affected side. Later this side may be wasted and deeply grooved—lingual hemiatrophy. Tremor of the tongue may be due to nervousness, thyrotoxicosis, delirium tremens, dementia paralytica or Parkinsonism.

Next examine the surface of the tongue and note (1) its colour. Is it pale, red or discoloured? Pallor is seen in severe anaemia. A slightly swollen, magenta-coloured tongue is found in riboflavin deficiency. (2) Is it dry or moist? The state of the tongue is a valuable indication of the state of hydration of the body. A dry brown tongue may be found in the later stages of any severe illness, but is found particularly in the later stages of uraemia and acute intestinal obstruction. (3) The presence or absence of fur. Furring of the tongue is of little value as an indication of disease. It is found in heavy smokers, mouth breathers, the edentulous, and those on soft, milky or otherwise sloppy diets. A brown fur—'the black hairy tongue'—is due to a fungus infection and is of no special significance, though frequently a source of great alarm to its possessor (Plate VI). The tongue gives no useful information about the state of the bowels. The tongue of scarlet fever at first shows bright red papillae standing out of a thick white fur. Later the white coat disappears, leaving enlarged papillae on a bright red surface—the 'strawberry tongue'. (4) The character of the papillae. Generalized atrophy of the papillae produces a smooth or bald tongue which is characteristic of pernicious anaemia, but may also sometimes be found in iron-deficiency anaemia, sprue, other gastro-intestinal disorders and deficiency states, especially pellagra. In severe cases smoothness may be associated with wrinkling of the mucous membrane, which has then to be distinguished from fissuring of the tongue.

seen in chronic superficial glossitis due to syphilis, and congenital fissuring of the tongue or 'scrotal tongue', which is of no pathological significance. In chronic superficial glossitis, areas of leukoplakia—whitish opaque areas of thickened epithelium—are separated by intervening smooth and scarred areas and no normal papillae are seen. In congenital fissuring (Plate VII) the papillae are normal, but the surface is interrupted by numerous irregular but more or less symmetrical furrows. In median rhomboid glossitis a lozenge shaped area of loss of papillae and fissuring is seen in the mid-line anterior to the foramen caecum (Plate VII). It may be mistaken for a carcinoma, and must be distinguished from a lingual thyroid which appears posterior to the foramen caecum. 'Geographical tongue' is another harmless anomaly in which the tongue shows localized smooth areas, which change in distribution (Plate VI). The 'false geographical tongue' with a similar appearance occurs temporarily in children with fever (Plate VI). (5) The undersurface of the tongue—a small ulcer on the fraenum is sometimes seen in persistent coughing, and particularly in whooping-cough. Sub-lingual varicosities are common in the elderly.

Inspect the mucous membrane on the inside of the cheeks. In the catarrhal stage of measles, before the appearance of the rash, small bluish-white spots, surrounded by a red areola, may be seen opposite the molar teeth. These are known as Köplik's spots. In the same situation irregular areas of slaty grey or blue pigmentation are seen in Addison's disease.

*Thrush* may sometimes be seen on the surface of the buccal mucous membrane, especially in children and in some patients being treated with antibiotics and is common under unclean dentures. It presents the appearance of small white points or patches raised somewhat above the surrounding surface, which is sometimes redder than normal. Patches of thrush are apt to be mistaken for small milk curds, but curds can be easily detached, while thrush patches can only be removed with difficulty, and when removed tend to leave behind a raw surface. To search for the fungus (*Candida albicans*) a small piece of the patch should be scraped off and examined in a drop of glycerin. A quantity of epithelial debris, along with bacteria and leucocytes, will be seen, and mixed up with these the filaments of the fungus. These consist of long but unequal segments usually possessing a refractile nucleus at each end.

#### The Palate, Fauces, and Pharynx

Introduce a tongue depressor and note first the general colour of

the soft palate, fauces and pharynx; observe any abnormal degree of pallor or redness. Remember that great insensitivity of the palate and pharynx is common in hysterical patients. Again, look for any ulcers or mucous patches on the palate, fauces or tonsils. The commonest ulcer seen in the mouth is a small, extremely painful, superficial one with a yellow-grey floor and bright red edges—the aphthous ulcer. Mucous patches are slightly-raised round or oval areas, covered by pearly-grey membrane. They are found in secondary syphilis, as are also superficial, circinate, 'snail-track' ulcers. Deep clean-cut ulcers with a thick felted grey-green slough on the floor and a characteristic odour occur in Vincent's angina. Large ragged ulcers or sloughs are seen in agranulocytosis and leukaemia. Instruct the patient to make the sound 'Ah' thus raising the soft palate and increasing visibility. Look carefully at the *tonsils*, noting any enlargement. Yellowish or greyish points or patches may sometimes be seen on their surface. See if these can be wiped off, leaving a sound surface, as is the case with accumulated follicular secretion, or whether removal leaves a raw surface, as happens with the false membrane of diphtheria. Note also if the soft palate and uvula show any similar spots or patches. The membrane of diphtheria is found characteristically on the mucous membrane of the fauces, as well as on the tonsils. Bacteriological examination of a throat swab (p. 355) is essential when there is a suspicion of diphtheria. In glandular fever reddish spots may sometimes be seen on the soft palate. Next look at the *pharynx*. The presence upon its surface of a number of flat adenoid swellings, somewhat like sago grains, is so common as to be almost a normal appearance. In chronic pharyngitis these are much increased. A few dilated venules can also be frequently observed. Notice any pus or excess of mucus on the surface and the existence of any ulceration. In retropharyngeal abscess the posterior wall of the pharynx is bulged inwards. Sometimes this can be more easily made out by palpation.

### The Breath

Bad teeth, gum or mucous-membrane ulceration, and retention and decomposition of secretion in the follicles of enlarged tonsils are the commonest sources of offensiveness in the mouth.

It is important to learn to recognize the characteristic odours of diabetes, uraemia and hepatic failure. In diabetic ketosis the breath smells of acetone, in uraemia there is a fishy or ammoniacal odour, and the *fetor hepaticus* has been described as 'mousy'.

In suppurative conditions of the lung the breath may have a putrid

smell, which may be elicited by asking the patient to cough. The odour of bronchiectasis has been compared to that of apple blossom with an *arrière-goût* of stale faeces. Paraldehyde also imparts its characteristic odour to the breath.

### The Oesophagus

#### Special Anatomy

The oesophagus is from 9 in. to 10 in. long. It begins opposite the cricoid cartilage, and ends opposite the 9th thoracic spine. It is crossed by the left bronchus between the 4th and 5th thoracic vertebrae.

In cases of *difficulty in swallowing* the radiologist must be asked to screen the patient and to report upon the course down the oesophagus of radio-opaque emulsion.

## II THE ABDOMEN

#### Anatomy

The *umbilicus* lies opposite the upper part of the 4th lumbar vertebra. Its position is far too variable for it to be a trustworthy landmark.

The *aorta* bifurcates about  $\frac{3}{4}$  in. below and slightly to the left of the umbilicus, the *iliac arteries* running in a line drawn from that point to a point midway between the anterior superior spine and the symphysis pubis.

The *coeliac axis* arises at a point  $4\frac{1}{2}$  in. to 5 in. above the umbilicus, and the *renal arteries* about an inch lower than the coeliac axis.

The *transpyloric plane* is often used as a guide in the examination of the abdomen. It is defined as lying midway between the suprasternal notch and the upper border of the symphysis pubis. It usually lies about halfway between the xiphisternal junction and the umbilicus, and it corresponds posteriorly with the lower border of the 1st lumbar vertebra.

#### General Examination of the Abdomen

The patient should be lying flat on his back in a good light. The abdomen is exposed by turning down all the bedclothes except the inner sheet. The clothing should then be drawn up and, lastly, the sheet folded down a little above the level of the pubes. These details are of special importance in examining female patients. Before beginning the examination of the abdomen, ensure that the bladder is

empty. Flexion of the hips is rarely of help. Nearly always the abdomen is best examined with the patient fully recumbent, with not more than one pillow and with the arms at the side.

### Inspection of Abdomen

First look at the general contour of the abdomen. Is it of normal fullness, is it swollen or protuberant, or is it sunken or retracted? If there is any bulging, note if it is general or local. General fullness may be due to 'fat, fluid, flatus, or faeces', to which aphorism may be added 'foetus' in the case of women. A *new growth* may also be a cause of general abdominal enlargement. In general bulging, it should be noted whether the distension is most marked in the antero-posterior or in the transverse diameter. In cases of general abdominal swelling measurement should be made at either the umbilical level or at the point of maximum distension. Observe in which zone local bulging is situated. Lastly, note if there is any movement to be seen in the swelling, either along with or independently of respiration.

*Pulsation in the epigastric region* may be noticed on abdominal inspection. The causes of it are, in order of frequency: (1) Aortic pulsation, which may be visible in any slightly-built patient with a thin abdominal wall. (2) Transmitted pulsation from a tumour (often a carcinoma of the stomach) overlying the aorta. (3) Distension of the right ventricle. (4) Venous pulsation of the liver. (5) Aneurysmal; the pulsation in this case is expansile.

The movements of the abdominal walls should be studied. Normally, they bulge during inspiration, and fall in during expiration. Paralysis of the diaphragm produces the opposite picture; sometimes the paralysis is unilateral, in which case one side of the abdomen will move naturally. Absence of movement of the abdominal walls is a valuable sign of peritonitis.

Intestinal obstruction produces tympanitic abdominal distension and sometimes visible peristaltic waves. The most extreme degrees of distension are seen in obstruction of the distal parts of the large intestine and chiefly affect the periphery of the abdomen. In obstruction at the ileo-caecal valve, the distended coils of the small intestine tend to stand out in the centre of the abdomen in a 'ladder pattern', but small intestinal peristalsis may sometimes be seen in old persons with thin abdominal walls and in the presence of divarication of the recti in persons who are otherwise healthy. In pyloric obstruction, the dilated stomach forms a prominent swelling in the upper part of the abdomen, and on gently shaking the patient a splashing noise is

produced (which may also occur in healthy patients after a meal, and in other forms of intestinal obstruction). Peristaltic waves in the stomach run from left to right. They are of particular importance in congenital pyloric stenosis of infants, when they may be the only diagnostic sign. Visible peristalsis may be easier to see if the infant is given a bottle or an adult a drink of soda-water.

Attention should next be paid to the *surface of the abdomen*. In great distension the surface is smooth and glossy. *Striae* (white or lilac lines in the epidermis) should be sought; they indicate a recent change in the size of the abdomen, and thus are found in pregnancy, ascites and wasting diseases. Conspicuous purplish striae are characteristic of the basophilism syndrome, but minor degrees may sometimes occur in obese people who are otherwise healthy. Note any *distension of the surface veins*, and endeavour to ascertain in what direction the blood is flowing. In obstruction of the inferior vena cava (Plate VIII) the inferior epigastric veins are full from the establishment of a collateral circulation. In such cases also a large *lateral vein* can be seen running up about the midaxillary line, establishing a communication with the tributaries of the superior vena cava. Distended veins may also be seen on the abdomen in portal obstruction (Plate VIII). Very rarely in this condition a number of distended veins may be seen radiating from the umbilicus. To this appearance the term 'caput Medusae' has been applied. It is due to establishment of a connection between the portal and parietal veins by means of the round ligament. Small distended venules in the region of the costal margin are of no significance. *Pigmentation* of the abdominal wall is sometimes important. Along the middle line it forms the linea nigra—one of the signs of pregnancy. Note the appearance of the *umbilicus*. Is it depressed, level with the surface, bulging, or everted, as in gross ascites? Lastly, one should never omit to look at the usual sites for any evidence of hernia.

#### Palpation of the Abdomen

The patient should lie flat on his back. He should be told to keep the mouth open and to breathe quietly; his attention may be diverted by conversation. The observer must sit or kneel beside the patient, in order to get his forearm into the horizontal position. Ordinary palpation is performed with one hand only. The hand must be warm. In order to gain the confidence of the patient, the hand should be allowed to rest for a moment on the surface of the abdomen before palpation is actually begun. Poking with the finger-tips should be avoided, the

best movement being a gentle one from the metacarpo-phalangeal joints, with the hand flat on the abdominal wall. Organs and masses in the upper abdomen are best felt against the radial border of the index finger as they descend in inspiration, at which time the hand may be moved gently upwards, as if to 'meet' them. To examine lateral regions of the abdomen, bimanual palpation is carried out. The physician sits or kneels by the bedside. One hand is placed posteriorly in the interspace between the last rib and the crest of the ilium. The other is placed over the abdominal wall in front. The posterior wall is then pushed up against the hand in front, so that any structure lying between the two hands can be distinctly felt. The secret of the method consists in keeping the front hand as still as possible. This procedure is of special value in the examination of the kidneys.

Begin always by a systematic very light palpation of the whole abdomen, noting any local or general rigidity or any marked tenderness. In this way the patient's confidence is gained and the later deep palpation rendered easier. It is wise to ask the patient whether any area is tender and, if so, to come to that area last. Normally, the abdomen has an elastic or doughy feeling only to be learnt by experience. In disease the resistance may be increased. General peritonitis produces rigidity from a reflex contraction of the muscles of the abdominal wall. Local rigidity is frequently due to localized peritonitis and is of great diagnostic value. Palpation of the normal abdomen is painless. If tenderness is elicited, its exact extent and point of maximum intensity should be noted. Tumours should be carefully felt for and care taken that a bulging rectus muscle does not cause confusion. The outline of the rectus muscle will become obvious if the patient is asked to raise his head against resistance.

If a tumour is really present, determine whether it is situated inside the abdomen or in the abdominal wall. Try to move the abdominal wall from side to side over the tumour. If the growth is intra-abdominal, this can usually be done without difficulty, unless it has become adherent to the parietal peritoneum. Try also to grasp the tumour and to make the fingers meet, as it were, under it. This can usually be accomplished in the case of tumours situated wholly in the abdominal wall.

If the tumour is intra-abdominal one must decide where it is growing from and especially whether it is coming up out of the pelvis or is truly abdominal. To decide this, place the hand about one inch below the umbilicus and push backwards and downwards in the direction of the sacral promontory. One can then feel whether the

tumour is passing down into the pelvis or not. The size and shape of the tumour should be noted, and the nature of its surface—whether smooth or nodular.

Tumours connected with the liver and spleen move freely with respiration, and so may those of the stomach. Tumours of the kidney may be slightly movable. Those connected with the other abdominal organs do not move with respiration.

### Percussion of the Abdomen

This should be carried out in the same manner as will be described for the chest, but particular care should be taken to percuss *lightly*. Percussion of the normal abdomen yields a tympanitic note throughout, except in the regions of liver and splenic dullness, or over a full bladder. Enlargement of these organs may sometimes be confirmed by percussion. The splenic dullness may be increased when the spleen is ruptured. The liver dullness is absent when there is gas or air in the peritoneal cavity. Such absence is a sign of perforation of a viscus, usually of perforation of a gastric or duodenal ulcer. The liver dullness is reduced in severe emphysema and in the presence of a right pneumothorax.

Free fluid in the peritoneum (*ascites*) is distinguished by the fact that it shifts its position with that of the patient. If he is turned over on his side and time given for the intestines to float up, the uppermost flank becomes resonant, while the height of the dullness on the lower side rises. This phenomenon is known as 'shifting dullness'.

The '*fluid thrill*' is another physical sign of fluid in the peritoneum. To elicit this sign, the patient is laid on his back, one hand is placed over the lumbar region on one side and the opposite side is flicked or tapped with the fingers of the other hand. A distinct impact will be felt to pass from one hand to the other. A similar impulse can be transmitted through a fat abdominal wall and so it is necessary for an assistant to place the edge of his hand firmly in the middle line of the abdomen while the tapping is performed. This damps down vibrations transmitted by the wall. On the whole the results of simple percussion afford the best evidence of the presence of ascites, but even this may be misleading. A fluid thrill can only be expected when the amount of fluid is large and it is under tension.

*Fat* is distinguished by taking the abdominal wall between the hands and pinching it up; *gas* by the results of percussion. Of *new growths*, ovarian cyst is probably the most liable to be mistaken for ascites. An ovarian tumour, however, causes an antero-posterior bulging of the

abdomen, while in ascites the bulging is mainly lateral. In ovarian tumours the dullness is central and does not change with the position of the patient: in ascites the chief dullness is in the flanks and it shifts when the patient is moved: the umbilicus is flat or bulges out in ascites, while in ovarian tumours it is drawn upwards: the 'slit' in the umbilicus is usually transverse in ascites and vertical in ovarian tumour.

### Auscultation of the Abdomen

Normally on auscultation of the abdomen numerous borborygmi are audible. However, in cases of general peritonitis or of intestinal ileus these sounds may be entirely absent and the 'silent abdomen' is a valuable physical sign. Excessive sounds are heard in intestinal obstruction.

## III THE ABDOMINAL VISCERA

### The Stomach

#### Special Anatomy

The normal stomach in the living subject is shaped like the letter J. The cardiac orifice usually lies on the left side of the 11th thoracic vertebra, and 4 in. behind the 7th left costal cartilage, 1 in. from the left border of the sternum. The position of the pylorus is at, or just to the right of, the mid-line and midway between the infrasternal notch and the umbilicus. It is normally under cover of the liver. The fundus of the stomach may reach as high as the 5th interspace in the midclavicular line, and rise a little above and behind the cardiac impulse. Only a small part of the body of the stomach and of the pyoric region is in contact with the anterior abdominal wall. The exact position of the greater curvature varies greatly according to the degree of distension of the stomach and the posture of the patient; radiographic examination has shown that it may be found anywhere from the region of the epigastrium and left hypochondrium to well below the brim of the pelvis.

### The Liver

#### Special Anatomy

The liver lies chiefly in the right hypochondrium. Its left lobe extends across the epigastric region, but does not pass more than 2 in. to the left of the sternum. Above, the liver reaches almost to the nipple; below, it extends to the costal margin. The lower border passes obliquely upwards from the 9th right to the 8th left costal cartilage,

crossing the mid-line somewhat above the mid-point between the base of the xiphoid and the umbilicus.

The gall-bladder is situated just internally to the 9th right costal cartilage, and immediately to the outer side of the right rectus muscle.

### Palpation of the Liver

Feel for the lower edge. To do this, place the hand flat on the abdomen, its edge towards the costal margin and to the outer side of the rectus muscle, thus avoiding the upper septum of the rectus sheath, which may be mistaken for the lower edge of the liver. Depress the edge of the hand slightly so as to push up a fold of skin and ask the patient to take a long breath. If the edge of the liver is palpable, it will be felt to ride under the edge of the hand. Trial must be made at different levels before it is decided that the edge cannot be felt. The edge of the liver can sometimes be felt in health and is frequently palpable in patients with emphysema. It moves down with inspiration. The character of the edge should be noted—whether it is smooth or irregular, soft or hard.

The *surface of the liver* in the epigastrium should then be felt in the usual way. Any tenderness should be noted, and whether it is localized or general. The character of the surface should be made out; whether it is soft, smooth and tender, as in heart failure; firm and finely nodular, as in portal cirrhosis; or hard and coarsely irregular, as in secondary carcinoma. Be careful not to confound little irregularities which are frequently present in the upper parts of the recti with irregularities on the surface of the liver.

### Percussion of the Liver

Use fairly heavy percussion. Begin high up at about the 2nd rib, to get a good lung note, and percuss down until impairment is detected. The upper limit of liver dullness forms an almost horizontal line around the chest. To define the lower edge of the liver, use very light percussion and pass upwards.

The *gall-bladder* cannot be felt unless distended, when it may form a smooth pear-shaped swelling, situated just to the outer edge of the right rectus muscle. It can be moved freely from side to side round a point opposite to the 9th costal cartilage. It moves with respiration.

If present, tenderness of the gall-bladder can be elicited by placing the hand beneath the costal margin in the right hypochondrium. The patient is told to take a deep breath and at the same time the hand is moved upwards underneath the costal margin. As the diaphragm

descends, the gall-bladder is driven against the fingers and if it is tender the breath is at once arrested with a gasp. This is spoken of as Murphy's sign.)

### The Spleen

#### Special Anatomy

The spleen lies in the left hypochondrium. It is bounded above and laterally by lung, medially by stomach and intestine. Its lower end rests upon the phrenicocolic fold of peritoneum. It lies along the 9th, 10th and 11th ribs, being partially separated from them by the diaphragm and lower edge of the left lung. Its upper end is opposite the 9th thoracic spine and is about  $1\frac{1}{2}$  in. from the middle line. Its lower end comes as far forward as the midaxillary line.

#### Palpation of the Spleen

The normal spleen is not palpable. A palpable spleen is enlarged, and it is never safe to diagnose enlargement of the spleen unless it is palpable.

Feel for the spleen standing on the right side of the patient. Place the flat of the left hand over the edge of the costal margin and firmly bring the ribs and lateral abdominal wall medially as the patient breathes in. Then start palpating with the right hand. (Large spleens are missed by starting palpation too near the costal margin.) The edge of the enlarged organ will be felt against the fingers of the right hand. Sometimes in the case of minor degrees of splenomegaly, the organ is best felt if the patient rolls over, half on to his right side towards the examiner.

The edge of the spleen is sharp and usually quite smooth. A notch can often be felt in it, but not invariably. It is important to note that the spleen enlarges downwards and to the right towards the right iliac fossa (Fig. 3), and that the anterior border of an enlarged spleen is always directed downwards and inwards.

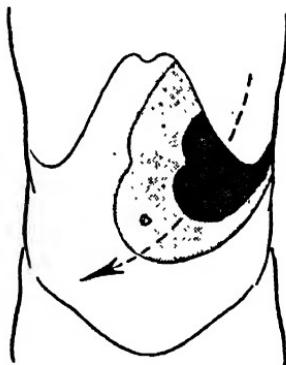


Fig. 3. Diagram to show direction of enlargement of the spleen.

### Auscultation

Auscultation over the spleen may be practised to detect the existence of friction. This occurs in perisplenitis and over the surface of splenic infarcts.

## The Kidneys

### Special Anatomy

Each kidney lies partly in the epigastric and partly in the hypochondriac region. The right kidney lies partly in the lumbar region as well. The kidneys are higher in relation to the anterior abdominal wall than is sometimes thought. The lower end of the right kidney is 1 in. above the umbilicus, the left about  $\frac{1}{2}$  in. higher. The lower end of each is about 3 in. from the middle line.

Posteriorly, about one-third of each kidney lies above the last rib. The upper end of the right kidney is at the level of the 11th thoracic spine, whilst its lower end is about 1 in. above the iliac crest. The left kidney is about  $\frac{1}{2}$  in. higher.

### Palpation of the Kidneys

The patient must lie on his back. The lumbar region must be flat and not arched forward. Sit or kneel beside the patient. Place one hand upon and below the last rib behind, the other immediately below the costal margin in front. The posterior hand should press the loin forwards, while the other hand pushes the anterior abdominal wall backwards, upwards and inwards. The kidney will then be felt between the two hands if it is at all enlarged or displaced. Even in health (if the patient is not too fat) the lower part of the organ can often be felt.

The kidneys move slightly with respiration. In health the lower pole of the right kidney is frequently palpable in persons of spare build, the left seldom so.

A movable right kidney is apt to be mistaken for a distended gall-bladder and vice versa. One point of distinction is that a distended gall-bladder can be temporarily pushed back from the abdominal wall, but always tends to spring forward again; it is therefore always in evidence. A movable kidney, however, often disappears for a time, and can only with difficulty be found again. Further, only the kidney is bimanually palpable.

An enlarged left kidney may be mistaken for the spleen. The

points of distinction are: (1) A renal tumour is usually 'bimanually palpable', that is, can be moved backwards and forwards between one hand in the loin behind and the other on the anterior abdominal wall. An enlarged spleen is not 'bimanually palpable'. (2) The fingers can usually be passed between the upper end of a kidney tumour and the ribs but not between the ribs and a splenic tumour. (3) The spleen has a sharp edge in which a notch can often be felt. The edge of the kidney is *always* rounded and has no notch.

An enlarged kidney tends to bulge forwards. Perinephric abscesses bulge backwards.

### The Intestines

#### Special Anatomy

The ileum joins the colon at a point 2 in. internal to, and somewhat above, the right anterior superior iliac spine. The base of the vermiform appendix usually lies opposite a point 1½ to 2 in. from the anterior superior spine along a line drawn from that spine to the umbilicus. This is sometimes called *McBurney's point*, because he showed that in the majority of cases of appendicitis it is the point of maximum tenderness.

The splenic flexure of the colon lies behind the stomach, the hepatic lies under cover of the liver. The former is at a higher level than the latter. The transverse colon passes across the abdomen in a curved direction. Radiography has shown that its position is very variable.

Examination of the intestines by *inspection* and *palpation* has been described under the general examination of the abdomen.

#### Rectal Examination

Place the patient in a good light and curled up on his left side or in a semi-prone position—i.e. resting on the left breast with the right thigh and knee well drawn up, the inner aspect of the right knee resting on the couch. Draw aside the buttocks and inspect the region of the anus, noting the presence of any eruption, of external haemorrhoids, etc. Fit a finger-stall to the right forefinger and smear it with petroleum jelly. If no finger-stall is available, fill the nail with soap and smear the finger with petroleum jelly. Massage the anus for a moment with the finger and then press gently with the pulp of the finger till it enters the anus, directing it slightly forwards at first.

Once the anal canal is passed, direct the finger slightly backwards and upwards, asking the patient to bear down a little at the same time.

The finger can then be swept round and the whole inner surface of the rectum explored.

In the male the prostate will be felt projecting into the rectum, and above it the trigone of the bladder flanked by the seminal vesicles; below is the membranous urethra. In the female the cervix will be felt projecting back in the form of a firm rounded swelling. Feel the mucous membrane for polypi, ulcers and malignant neoplasm.

Remember that haemorrhoids are not palpable unless they are thrombosed. The presence of scybala or foreign bodies can be determined. If the lymphatic glands which lie in the hollow of the sacrum are enlarged, they may be felt. If secondary malignant deposits or an abscess are present in the recto-vesical pouch, the mass will be palpable through the wall of the rectum. On withdrawing the finger, examine the finger-stall for the presence of mucus, blood or melaena. Fragments of stool adhering to the finger-stall can be tested for occult blood (p. 57).

### Proctoscopy and Sigmoidoscopy

If rectal examination is negative and there is reason to suspect abnormality near the anus, the anal canal and lower three inches of the rectum should be examined with the proctoscope. Place the patient in the position described for rectal examination, and pass the warmed, lubricated instrument carefully to its full depth. Remove the obturator and inspect the mucous membrane as the instrument is slowly withdrawn. In this manner haemorrhoids may be seen or the nature of a palpable abnormality directly ascertained.

It is often necessary to examine the rectum and colon more fully than is possible by proctoscopy, and in such cases the sigmoidoscope is employed. Sigmoidoscopy requires skill and experience. In accomplished hands the instrument can be passed for at least 20 cm. and a further 4 cm. or so of the colon is visible beyond this. The procedure causes very little discomfort and anaesthesia is unnecessary and undesirable.

Sigmoidoscopy is particularly useful in the differential diagnosis of diarrhoea of colonic origin. It serves to distinguish carcinoma of the colon, the ulcerated bleeding mucosa of ulcerative colitis, polypi in polyposis coli, and the red granular surface in granular proctitis. In suspected amoebic dysentery, the mucous membrane may be inspected, and portions of mucus or scrapings from ulcers may be removed, mounted in saline on slides, and examined microscopically for amoebae and cysts. When the sigmoidoscope is used for this purpose, it

should be lubricated with a non-greasy preparation, such as mucilage, or droplets of oil will interfere with the microscopical examination.

### Aspiration of Peritoneal Fluid

This is undertaken for diagnostic and therapeutic purposes. It is essential first to make sure that the bladder is empty, and if there is any doubt a catheter should be passed before the aspiration is attempted.

The patient should be lying flat or propped up at an angle of 45°. A binder or many-tailed bandage should be placed in position around the patient's back before the aspiration is begun. The aspiration can be made in the mid-line half-way between the umbilicus and the symphysis pubis; but is more often done in the right iliac fossa, a little outside the mid point of a line drawn from the umbilicus to the anterior superior spine.

With suitably sterile precautions, the skin at the point chosen should be infiltrated with local anaesthetic, and then the anaesthetic should be injected down to the parietal peritoneum. If the puncture is made simply for diagnostic purposes, a 10 ml. syringe and a suitable needle can be used. If, as is more usual, it is intended to drain the peritoneum, a trochar and flanged cannula (which can be fixed to the skin with adhesive tape) should be employed. It is wise to tie the rubber tubing (which is used to drain the fluid into a bottle) on to the flanged canula, then to stretch the tubing over the end of the cannula and to insert the trochar through the rubber tubing. This saves soiling of the bedclothes when the trochar is withdrawn.

A tiny incision should be made in the anaesthetized area of skin, and then the trochar and cannula should be inserted. A resistance is felt as the trochar perforates the parietel peritoneum. The trochar should then be withdrawn, and the fluid should drain into the bottle. The rate of flow, which should not be too fast, can be controlled by means of a clip on the rubber tubing. When the aspiration is complete, the cannula should be withdrawn. The puncture can be sealed with collodion and a dry dressing applied.

The fluid can be examined bacteriologically, chemically and for cytology. Transudates such as occur in heart failure, cirrhosis and nephrosis normally have a specific gravity less than 1018 and a protein content under 2.5 g. per litre. Exudates, occurring in tuberculous peritonitis or in the presence of secondary deposits, usually have a specific gravity above 1018 and more than 2.5 g. of protein per litre. The distinction, however, is somewhat unreliable. Tubercl

bacilli may be demonstrated in the fluid in tuberculous peritonitis; and a blood-stained fluid strongly suggests metastases. Malignant cells may also be demonstrated in the latter condition.

#### IV SPECIAL INVESTIGATIONS OF THE ALIMENTARY AND BILIARY TRACTS

Under this heading methods of examining the stomach, gall-bladder and intestine by intubation and radiology will be described.

##### Intubation

The original rubber Ryle's tube has been replaced by plastic tubing, which is less irritant and easier to sterilize than the rubber variety. The tubes are sterilized by gamma radiation and supplied in sealed packs. The tube is weighted with metal at the blind end, and there are a number of small perforations 2 cm. from the tip. Along the tube are a number of printed lines, each indicating the position of the tip of the tube when the mark is opposite the teeth, throat, oesophagus, cardia, stomach and duodenum. These marks, however, are valid only when the tube is straight and not coiled up either in the mouth or in the body of the stomach.

All tests in which a gastric tube is passed leave a 20 per cent chance of major error unless the position of the tip is checked radiologically. The tube must lie at the angulus. If it is coiled at the fundus, a false impression of achlorhydria may be obtained.

The tube is placed unlubricated on the back of the patient's tongue, and he is asked to close his mouth and swallow the bulbous end just like a pill. Swallowing is continued till the appropriate mark almost reaches the teeth. The position of the tip of the tube in the stomach is best confirmed by radiography, as it sometimes curls up in the fundus. When necessary, the tube can usually be induced to pass on into the duodenum. The patient should lie on his right side, preferably with his pelvis elevated on pillows. Usually within an hour the bulb passes into the duodenum, as indicated by the fact that the fluid withdrawn is alkaline to litmus or consists of frank bile. Again the position of the bulb is better confirmed radiographically.

Intubation is now more used for therapeutic than for diagnostic purposes. It is worth remembering, however, that in extensive carcinoma of the stomach, the fasting-stomach juice is usually a cloudy, foul-smelling fluid containing débris and having a pH of from 4 to 6. Some physicians leave a tube in the stomach of patients with

upper gastro-intestinal bleeding, to assist in determining when bleeding has stopped or when fresh bleeding occurs.

## 2 Test Meals

The fractional test meal described in previous editions has been abandoned by most physicians as an aid to the diagnosis of dyspepsia. Its main disadvantage was that it measured only the acidity of samples of gastric contents, which depends as much on the rate of gastric emptying and on the dilution of the stomach contents by saliva and regurgitated duodenal juices as on gastric secretion. There is as yet no method of measuring gastric secretion which entirely avoids this difficulty. At the moment, test meals are used mainly for two purposes —to detect achlorhydria in pernicious anaemia, and to detect gross hypersecretion in the very rare Ellison-Zollinger syndrome. Some surgeons use them as an aid to deciding the type of operation to be performed in peptic ulceration. For these purposes, either the histamine test meal or the augmented histamine test meal may be used. In both tests continuous aspiration of stomach contents by means of an electric pump is recommended, but periodic aspiration every 10 to 15 minutes with a 20-ml. syringe may be employed. A radiopaque Levine's tube (larger than Ryle's) should be employed, and it should be demonstrated radiographically that the tip lies in the pyloric portion of the stomach.

In the histamine test meal, the subject is fasted for 12 hours and 0.5 mg. of histamine acid phosphate is injected subcutaneously. The gastric juice is then aspirated for 60 minutes. The normal secretion is up to 200 ml. in 60 minutes, with an acidity equivalent to 180 ml. of decinormal hydrochloric acid.

In the augmented histamine test meal, a large dose of histamine (0.04 mg. per kilo of body weight) is given with the object of causing maximum secretion and so obtaining an indication of maximum secretory capacity or 'parietal cell mass'. All actions of histamine other than that on the parietal cells are neutralized by a suitable dose of antihistamine. The procedure is as follows. After a 12-hour fast, the gastric contents secreted spontaneously are aspirated continuously for 1 hour. The patient is then given an appropriate dose of antihistamine (usually 50 mg. of mepyramine by intramuscular injection). The juice aspirated in the next half-hour is discarded, and at the end of this time 0.04 mg. of histamine acid phosphate per kilo of body weight is injected subcutaneously. The juice secreted for the next hour after this injection is then collected.

The average normal basal secretion is 0·3 milliequivalent HC1 in 1 hour and the average normal maximum output is less than 22 milliequivalents in 1 hour (male) and less than 15 milliequivalents in 1 hour (female).

The figures given as normal in both these tests are considerably exceeded in duodenal ulcer and enormously exceeded in the rare Ellison-Zollinger syndrome, where the volume of juice secreted is also greatly increased. Achlorhydria has traditionally been regarded as a pH of more than 3·5 (turning point of Topfer's reagent). But some acid must have been secreted for the stomach contents to reach a pH of 3·5. It has therefore been suggested that for the diagnosis of pernicious anaemia, the acidity of the gastric contents should be expressed in terms of pH and that achlorhydria should be defined as a failure to lower the pH beyond 6·0 in response to maximal histamine stimulation.

For the diagnosis of dyspepsia accurate radiology is of far more value than any form of test meal.

### 3 Small-intestinal Biopsy

A safe and simple technique for biopsy of the small intestinal mucosa employs a spring-loaded capsule (the Crosby capsule). This capsule is about 1·5 cm. long and 7 mm. in diameter. It contains a cutting-blade which excises a small piece of intestinal mucosa when negative suction is applied to the thin plastic tube to which it is attached. The capsule is swallowed, and a small amount of radiopaque dye is injected, to check that it has passed through the pylorus. After the biopsy has been taken, the capsule is pulled up and the specimen placed on a small piece of card in a preservative solution. It should be examined immediately by the pathologist under a low-power microscope, to assess the macroscopic appearance of the villi.

This procedure must always be preceded by a barium meal and follow-through examination, as the technique might be dangerous if the capsule were to lodge in a duodenal or jejunal diverticulum.

Small-intestinal biopsy is of particular importance in the diagnosis of the malabsorption syndrome, where a flat mucosa is seen in place of the usual multiple villi.

### 4 The Barium Meal, Follow-through Meal and Barium Enema

These radiological methods are often of the greatest value, but for details of their performance the student must consult special textbooks.

For the barium meal the patient swallows a suspension of radiopaque barium sulphate, while the radiologist observes its passage on

the fluorescent screen, or on the TV monitor screen of an image-intensifier. Films are taken to provide a permanent record of any abnormality discovered, but, as in all barium examinations of the alimentary tract, screening is important. The barium meal is principally used in the diagnosis of gastric and duodenal ulcer and of gastric carcinoma.

- . In *gastric ulcer* the radiologist may demonstrate:
  - (i) an ulcer crater as indicated by a projection of barium or by a characteristic distortion of the mucosal pattern;
  - (ii) a localized spasm of the stomach opposite the ulcer, known as an 'incisura'.

- In *duodenal ulcer*:
- (i) an ulcer crater;
  - (ii) deformities of the duodenal cap due to spasm or scarring which produce a 'trefoil' appearance;
  - (iii) an increase in resting-juice, associated with pyloric obstruction;
  - (iv) an 'irritable cap' with very rapid passage of the barium from the stomach to the second part of the duodenum, making the duodenal bulb difficult to demonstrate.

In *gastric carcinoma* a portion of the stomach may be found, which cannot by any manipulation be filled with barium, and which remains constant in outline. It is known as a 'filling defect'.

Evidence may also be found of complications such as hour-glass constriction of the stomach due to ulcer or pyloric obstruction due to ulcer or carcinoma.

The small intestine may be studied by taking films of the abdomen at intervals after the barium meal. Abnormalities in small-gut pattern—widening, narrowing, increase in transverse barring or flocculation—and in the transit-time to the colon, as well as the presence of fistulae, may be demonstrated, and thus a diagnosis of Crohn's disease, malabsorption syndrome, or neoplasm may be confirmed. Many radiologists prefer to give smaller quantities of a non-flocculating barium suspension, either by mouth or via a duodenal tube—the small bowel enema'.

In the *barium enema* the suspension is introduced into the rectum as an enema, and manipulated round the colon to the caecum. By this means, obstruction to the colon, neoplasm, diverticulosis, and other abnormalities can be recognized.

### 5 Cholecystography and Cholangiography

*Cholecystography* depends on the fact that certain iodine-containing

compounds are excreted by the liver and concentrated in the bile and so render the gall-bladder radiopaque.

The substance most used in oral cholecystography is Telepaque (iopanoic acid).

After a preliminary radiograph has been taken, to see if radiopaque gall-stones are present, a dose of the preparation suitable for the patient's weight is given by mouth after the patient's evening meal, which should be light and free from fat. Several radiographs are taken 12-16 hours later. If the gall-bladder is seen, the patient is given a fatty meal and its emptying is observed.

Failure to demonstrate the gall-bladder may be due to the fact that the dye was vomited or not absorbed on account of diarrhoea or other cause, that hepatic function is impaired—it is generally useless to attempt cholecystography in a jaundiced patient—or that disease of the gall-bladder is present. Deformities of the gall-bladder shadow due to structural changes and calculi, whether opaque or not to X-rays, may thus be demonstrated.

*Cholangiography* can be performed by the intravenous administration of Biligrafin (a compound of triiodo-benzoic acid). 20 ml. of 20 per cent Biligrafin is injected intravenously. A film is taken 20 minutes later and then serial films at 10-minute intervals. In successful cases the biliary ducts are outlined, so that patency of the ducts and the site of extrahepatic obstructions can be demonstrated. The method is particularly valuable to show strictures or calculi in patients whose gall-bladders have been removed. It will not, however, be successful in patients with more than slight degrees of jaundice. Increasing the dose of oral preparations does not usually produce denser shadows of the gall-bladder or ducts. Intravenous cholangiography is best performed immediately the cholecystogram has been found to fail, that is, sixteen hours after the oral Telepaque.

## 6 Gastroscopy

Gastroscopy is a specialized method of investigation requiring considerable experience. The technical procedure will not be described here. It is carried out under local anaesthesia, and although it may cause considerable discomfort to the patient, it is safe provided that no oesophageal abnormality is present. Effective inspection of the body of the stomach may be obtained, but the pylorus and cardia are less easily seen. Doubtful radiological appearances may be confirmed or refuted, and it may be possible to express an opinion as to the malignancy or otherwise of a gastric ulcer seen on X-ray examination.

### 7 Gastro-camera

This instrument is passed by mouth like a gastroscope, and allows photographs to be made of the mucosal surface of the stomach, which has been distended with air. A permanent record of the intra-gastric appearances is thus available. The latest fibroscopic instruments allow the area to be photographed to be seen first. They are sufficiently flexible and manoeuvrable to allow almost all the stomach to be visualized.

### 8 Liver-function Tests

The liver is remarkable for the number and variety of its functions. Numerous tests of individual functions have been used. No single test can give a composite picture of liver function. The more important tests are:

(1) Tests depending on failure to excrete bilirubin:

- (a) **Serum bilirubin.** See p. 372.
- (b) **Urobilinogen in urine.** See p. 183.

(2) Tests depending on metabolism of protein:

(a) **Plasma proteins.** The liver is mainly, if not wholly, responsible for the production of serum albumen, whereas globulin is mainly produced elsewhere. In advanced liver disease, particularly in cirrhosis, there is a considerable reduction in plasma albumen (normal 4.0 to 5.5 g. per 100 ml.) without a corresponding fall in globulin (normal 1.4 to 3.0 g. per 100 ml.), which may even be increased, and thus there is a fall in the albumen-globulin ratio. There are also widespread changes in the electrophoretic pattern.

(b) **Flocculation tests.** There are changes in the different globulin fractions in liver disease, which alter their electrophoretic pattern and are thought to be the basis of the flocculation tests. Positive results are found in many cases of active liver disease. The most commonly used are:

Thymol turbidity (normal 0-4 units).

Zinc sulphate turbidity (normal 0-8 units).

These tests are empirical and positive results may

## THE ALIMENTARY SYSTEM

occur in other diseases associated with hyperglobulin-aemia, e.g. infectious mononucleosis, multiple myeloma, rheumatoid arthritis, malaria and kala-azar.

(c) **Plasma prothrombin.** Since prothrombin is formed in the liver from vitamin K, the prothrombin index (p. 129) is reduced in obstructive jaundice, when vitamin K is not absorbed, and in advanced liver disease, when prothrombin is not formed in the liver. A low prothrombin index, persisting after injections of synthetic vitamin K (menaphthone), suggests severe impairment of liver functions.

(3) Test depending on excretory functions:

**Alkaline phosphatases.** These are enzymes which form inorganic phosphates from phosphoric acid esters. They are formed in bone and are excreted by the liver. Their concentration in the blood is increased in bone diseases, such as osteitis fibrosa, hyperparathyroidism (sometimes), bone neoplasms and rickets, and also in jaundice, particularly obstructive jaundice. The normal serum concentration is from 3 to 13 King-Armstrong units per 100 ml. in adults, and 10 to 30 units in children.

(4) Test depending on necrosis of liver cells:

**Serum transaminases.** These are enzymes which appear in the blood after tissue destruction. In active hepatitis, their serum level is usually considerably increased.

Liver function tests are employed:

(1) *to assist in the differential diagnosis between jaundice due to hepatitis and that due to extrahepatic biliary obstruction.* The diagnosis is made mainly upon consideration of the history and physical signs, but complementary tests are valuable. Absence of urobilinogen from the urine (p. 183) indicates complete obstruction. This rarely persists for long in hepatitis, and is usually intermittent in gall-stones. Thus, persistent complete obstruction, demonstrated by repeated negative urinary urobilinogen tests, favours obstructive jaundice due to growth.

In jaundice due to extra-hepatic obstruction, liver function is normal at first, and fails gradually. In acute hepatitis, liver dysfunction is maximum at first and usually lessens. Thus, in chronic jaundice repeated liver-function tests showing deterioration in

function suggest obstructive jaundice, while tests showing improvement of function suggest hepatitis.

The non-specific flocculation tests are positive early in jaundice due to hepatitis, but not in obstructive jaundice, whereas the alkaline phosphatase is raised early in obstructive jaundice but not in hepatitis. Thus, alkaline phosphatase levels above 40 units with negative or weakly positive flocculation tests indicate obstruction, while phosphatase levels below 15 with positive flocculation tests indicate hepatitis. The serum transaminase level is raised in hepatitis, but not in extrahepatic obstruction. Chlorpromazine and other drugs, however, may produce jaundice with liver-function tests identical with those of extrahepatic obstruction.

(2) *To detect liver failure, either to confirm a clinical diagnosis of hepatic disease or to estimate prognosis when operation is contemplated.* For this purpose several tests should be performed. In advanced cirrhosis and in hepatic failure from other causes, typical findings are a low plasma albumen (less than 4.0 g. per 100 ml.), a low prothrombin index, raised plasma bilirubin and a raised alkaline phosphatase level. The alkaline phosphatase may be raised—in the absence of jaundice—in primary carcinoma of the pancreas, and in the presence of hepatic secondaries from any source.

Reliance must not be placed exclusively upon the results of liver-function tests, which are often unhelpful and occasionally misleading.

## V EXAMINATION OF VOMIT

The character of the vomit varies with the nature of the food ingested and the absence or presence of bile. In *pyloric stenosis* the vomit is apt to be copious and sour-smelling, and exhibits a froth on the surface after standing. The presence of much *mucus* gives the vomit a viscid consistency. The appearance of the vomit in *haematemesis* varies. If the bleeding is copious, the vomit may present the appearance of pure blood and contain clots. Such bleeding may come from a gastric ulcer or from the oesophageal varices of portal obstruction. More commonly the blood is altered in colour by being in contact with the gastric juice; it may be blackish in colour, or dark brown. The latter appearance is due to the conversion of haemoglobin into haematin. The altered blood gives to the vomit an appearance often compared to that of *coffee-grounds*. The taking of preparations of iron or red wines may produce a similar appearance in the vomit. Vomit which contains dark-green bile may resemble vomit which contains

blood. On diluting with water, however, the green colour of the bile becomes more apparent, while blood remains dark. If there is any doubt, the tests for blood described under the examination of faeces may be applied. Remember that blood in vomit may have come from the nose or lungs and have been swallowed. Faecal vomit, characteristic of advanced *intestinal obstruction*, is brownish black in colour and may resemble altered blood, but has a typically faecal odour.

## VI EXAMINATION OF FAECES

Examination of the faeces is an investigation of great importance too frequently omitted. No patient with bowel disturbance has been properly examined until the stools have been inspected. The white surface of a bedpan makes an ideal background for the detection of blood, pus, and mucus.

### 1 Naked-eye Inspection

The following points should be noted:

The *amount*. It is sufficient to state whether the stools are copious or scanty.

The *colour*. *Black* stools may be produced by the ingestion of iron or bismuth. In haemorrhage occurring high up in the intestine the altered blood makes the stools dark, tarry-looking, and very offensive, and the guaiac test and Haematest are strongly positive. *Pallor* of the stools may be due to lack of entrance of bile into the intestine, as in obstructive jaundice; to dilution and rapid passage of the stool through the intestine as in diarrhoea; or to an abnormally high fat content, as in sprue.

The *odour*. The stools in jaundice are often very offensive. Cholera stools, on the other hand, contain very little organic matter, and are almost free from odour. The stools of acute bacillary dysentery are almost odourless, while those of amoebic dysentery have a characteristic odour, something like that of semen.

The *form* and *consistence*. In constipation they may be drier and harder than normal, and sometimes resemble sheep-stools. In all forms of diarrhoea they are more fluid than normal, and may be watery. Slimy stools are due to presence of an excess of mucus.

To detect *abnormal ingredients*, the stool should be placed on a fine sieve, and a large quantity of water added. The whole is shaken and stirred up till the soluble parts are all washed away: The residue is then examined. The head of a tapeworm can best be seen if this

residue is strained through black muslin. The head is about as large as that of a large pin, and the neck about as thick as a stout thread.

Watery stools are found in all cases of profuse diarrhoea, and after the administration of purgatives. To the stools of cholera the special name of *rice-water* stools is applied. Such a stool is colourless, almost devoid of odour, alkaline in reaction and contains a number of small flocculi consisting of shreds of epithelium and particles of mucus. Purulent or pus-containing stools are found in severe dysentery or ulcerative colitis, or in cases where an abscess has found its way into the intestines. Slimy stools are due to the presence of an excess of mucus, and point to an affection of the large bowel. The mucus may envelop the faecal masses, or may be intimately mixed with them. Bloody stools vary in appearance according to the site of the haemorrhage. If the bleeding takes place high up, the stools look like tar. In an intussusception they may look like red-currant jelly. If the haemorrhage is from the large intestine, the blood is less intimately mixed with the faecal matter, and may even be of a bright colour. In haemorrhage from the rectum or anus it may merely streak the faecal masses. The stools of bacillary dysentery consist at first of faecal material mixed with blood and pus, later of blood and pus without faecal material. Those of amoebic dysentery characteristically consist of fluid faecal material, mucus and small amounts of blood. The stools of steatorrhoea are very large, pale and putty-like or porridge-like and sometimes frothy.

In so-called muco-membranous colitis the motions may contain casts of the bowel, which are mainly composed of mucin. The individual casts vary considerably in size, being commonly from 1 in. to 6 in. long. These casts, when small, may, on casual observation, be mistaken for segments of tapeworm.

## 2 Chemical Examination

**Tests for occult haemorrhage.** Since benzidine ceased to be available, on account of a high incidence of carcinoma of the bladder in those who made it, orthotolidine has taken its place.

(1) *Orthotolidine test.* A portion of faeces the size of a pea is suspended in 5 ml. of distilled water and boiled for 5 minutes. A 4 per cent stock solution of orthotolidine in 95 per cent ethyl alcohol is prepared. For use a 1-in-5 solution is made in glacial acetic acid. 1 ml. of this reagent, 0.25 ml. of the faecal suspension, and 0.25 ml. of hydrogen peroxide (20 vol. strength) are mixed in a test-tube, and the result read in 3 minutes. A strong positive is indicated by a dark green colour and a weak positive by a pale green. This is a moderately sensitive test,

and to avoid false positives the patient should have been on a meat-free diet for 3 days before the test and should not have been taking aspirin.

(2) *Haematest tablet test.* These tablets contain orthotolidine and strontium peroxide. A blue colour is produced when a wetted tablet is in contact with faeces containing blood.

A thin smear of faeces is made on the test-paper provided. A tablet is placed in the centre of the smear, and two drops of water are run on to the tablet. The water overflows on to the smear of faeces. A slight blue colour on the tablet is of no significance, but a blue colour developing in the smear at the edge of the tablet within 2 minutes is a positive result.

This test is relatively insensitive. It can therefore be used on patients on a normal diet, but will not detect small amounts of gastro-intestinal bleeding.

Both tests are of value in indicating the presence of gastro-intestinal bleeding, but both may be negative in the presence of lesions which bleed intermittently or slightly, particularly those situated in the upper gastro-intestinal tract.

**Fats in faeces.** Fat is present in food as neutral fat or triglyceride. It is split to greater or lesser degree by lipases, mainly of the pancreas, into glycerol and fatty acids. Some of the fatty acids, if unabsorbed, combine with bases to form soaps. Fat may, therefore, be found in the faeces as neutral fat, fatty acids and soaps.

The estimation of the proportion of split and unsplit fats present has been found unreliable as a method of distinguishing pancreatic from non-pancreatic steatorrhoea.

For the estimation of the fat in the stools, the patient may be placed on a diet containing 50 g. of fat per day. The fat present in the stools collected over three or better five days is then estimated. It has been found that equally reliable results are obtained if the patient eats a normal diet. In these circumstances not more than 6 g. of fat should be excreted each day, and any figure in excess of this indicates steatorrhoea.

### 3 Microscopical Examination

See protozoa in faeces (p. 62).

## VII INTESTINAL PARASITES

The parasites which occur in the intestinal tract include worms and protozoa. Some of the nematode and cestode worms will be described.

### 1 Nematoda

Perhaps the commonest of all internal parasites is the threadworm, *Enterobius vermicularis*, whose presence is associated with considerable itching about the anus. It inhabits the large intestines, caecum and appendix, and female specimens can often be seen wriggling about in the recently passed motion of their host. To the naked eye they look like small white threads, 0·5 to 1 cm. in length. Under the microscope the female may be distinguished by her much larger size, by the large uterus filled with ova, and the pointed posterior end. For appearance of ovum, see Plate XII, 15.

*Ascaris lumbricoides*, or roundworm, has a general resemblance to an earthworm. It usually measures from 6 to 8 in. but may be up to 13 in. long. The ova, which can occasionally be found in the dejecta, have brownish-yellow granular contents, and in many cases the shell is surrounded by an irregular sheath (Plate XII, 18).

*Ankylostoma duodenale*, or hookworm, is an important cause of anaemia and debility in the tropics, where heavy infestations may occur. It is no longer indigenous in Great Britain, but may be found in immigrants. It lives for the most part in the upper part of the jejunum, and its presence there is probable when, in an infested district, severe anaemia, otherwise inexplicable, sets in. The diagnosis is confirmed by the discovery of ova in the motions (Plate XII, 14). They exhibit a segmented yolk enclosed in a thin shell, and are sufficiently numerous to be readily detected. The adult worm, which is rarely seen before therapeutic agents have been employed, is about half an inch long, and the mouth is provided with four claw-like teeth.

*Trichina spiralis* gains access to the body as the result of eating infested pork. Trichiniasis is rare when pork is eaten cooked, but small outbreaks and sporadic cases have been reported in Great Britain and in the USA. When man ingests the muscle trichinellae of the pig, larvae are set free in the small intestine, giving rise to the symptoms of the first stage of the illness—abdominal pain, vomiting, and diarrhoea. The adult female, 3 mm. long, penetrates the intestinal wall and discharges embryos into lymph spaces, whence they migrate into muscles. In this second stage of the illness the patient has fever and the muscles swell and become hard and tender. Rarely death may occur at the height of the myositis. Otherwise the embryo undergoes no further development, and its capsule becomes calcified. Unlike cysticerci, described below, calcified trichinellae are not usually visible in X-rays.

## 2 Cestoda

Many different kinds of tapeworm have been found as parasites in man, but the most important are *Taenia saginata*, *T. solium* and *T. echinococcus*. Besides its occurrence in the fully-developed state, *T. solium* may be present in the tissues in the form of a cysticercus; *T. saginata* is never found in this condition in man; whilst *T. echinococcus* always occurs in the cystic stage, and has never been found in the mature condition in the human intestinal tract.

The presence of an adult tapeworm in the bowel is generally revealed by the passage of ripe proglottides in the stools, and after the administration of anthelmintics, the head may be detected by the methods previously described (p. 56).

*Taenia saginata (mediocanellata)* is the beef tapeworm. Infestation occurs as a result of consuming insufficiently-cooked beef infested with the embryo of the worm. The adult parasite reaches a length of 4 to 8 metres and consists of about 2000 segments. The ripe proglottides measure 13 by 6 mm. The head is quadrate, measures 2 mm. in diameter, has four suckers, but is devoid of hooklets. The terminal gravid segments of the worm from time to time become separated and the ova are then ingested by the bullock or cow, in the muscles of which the larva develops. It becomes a bladder worm, *Cysticercus bovis*, measuring 10 by 6 mm. and containing an invaginated head which possesses in miniature the characteristics of the adult scolex. *Cysticercus bovis* is never found in human muscle tissue or brain. For appearance of ovum, see Plate XII, 16.

*Taenia solium*, the pork tapeworm, is not encountered in Britain but is endemic wherever infested pork is eaten raw or insufficiently cooked. It measures 2 to 4 metres in length; a ripe proglottis is 10 by 5 mm. The head measures 1 mm. in diameter and in addition to four suckers has a rostellum with 32 hooklets. The ova in the terminal proglottides are ingested by the pig, in the muscles of which the bladder worm *Cysticercus cellulosae* develops. Occasionally persons in infected areas become infested with *Cysticercus cellulosae* from eating food contaminated with the ova of the parasite. The muscles of the human host are then infested by cysticerci, which are palpable through the skin as tense ovoid swellings 10 by 5 mm. and of almost cartilaginous hardness. About four years after infestation they become calcified and may then be demonstrated radiologically. The thigh muscles are those in which cysticerci are most easily demonstrated. Cysticerci may also occur in the brain and are a cause of epilepsy.

*Taenia echinococcus.* The adult worm, which consists of a head and three segments, and whose length is only 3 to 8 mm., need not be fully described, since it is not found in man. The *cystic stage* is very important, as it gives rise to serious disease in man in many of the viscera and especially in the liver. The cysts of this taenia are not simple, but produce from their inner surface one or two generations of secondary vesicles, on which the brood-capsules, containing the cestode heads, are formed. During the period in which this process is going on, the primary vesicle dilates to accommodate its increasing contents, and may eventually reach the size of a coconut. The vesicles may rupture spontaneously, and their contents may escape by the lungs, by the bowel or by the urinary passages. Specimens may be obtained by aspiration or after surgical interference.

The diagnosis of suspected hydatid disease may rest upon the recognition of the nature of fluid withdrawn, of hooklets or scolices, or the appearance of parts of the ectocyst, which are sometimes coughed up from the lungs.

The fluid is clear, alkaline, devoid of protein, and contains abundance of sodium chloride and traces of glucose. Its density is low, being generally under 1010. The *scolex*, if it is obtained in a perfect condition, is about 1 to 1.5 mm. in diameter, and a number of them often spring in a group from one brood-capsule. They have four suckers and a crown of hooklets. Portions of the *ectocyst* appear as whitish-yellow shreds, which can be recognized under the microscope by their lamination and by the pectinate markings on the laminae.

*Dipyllobothrium latum* (*Dibothriocephalus latus*), the fish tape-worm, is encountered in Sweden, Finland and Michigan. The adult worm measures from 3 to 10 metres or more and has a total of 3000 segments. The scolex is small, spatula-shaped and possesses two deep suctorial grooves. The first larval host is a water-flea and the second the pike, perch or salmon trout. Human infestation takes place from eating raw or under-cooked fish. In a tiny proportion of cases, a vitamin B<sub>12</sub> deficiency anaemia may be produced.

### 3 Trematodes

Schistosomes or blood flukes are the most important trematode parasites of man. They are found in three varieties, and produce the disease known as schistosomiasis or bilharziasis. *Bilharzia japonicum* and *Bilharzia mansoni* inhabit the portal blood stream, and the ova (Plate XII, 20, 21) are passed in the faeces. *Bilharzia haematobium* characteristically inhabits the vesical plexus, so that the ova (Plate XII, 19) are passed in the urine (p. 188). They may occasionally also be

found in the faeces. *Bilharzia mansoni* is found in Africa and South America; *Bilharzia haematobium* in Africa and the Near East, particularly in Egypt; and *Bilharzia japonicum* in the Orient.

#### 4 Protozoa

A number of protozoa, many of them non-pathogenic, have been found in the faeces. Of these the most important clinically is *Entamoeba histolytica* (Plate XII, 1, 2 and 3), which causes amoebic dysentery (as opposed to bacillary dysentery) and sometimes tropical abscess of the liver. *Entamoeba coli* (Plate XII, 5, 6 and 7) is non-pathogenic.

*Entamoeba histolytica* is found in the stools in two forms. In acute attacks vegetative amoebae can generally be found, whilst in the more quiescent stage cysts are passed.

If amoebic dysentery is suspected, a stool should be passed into a clean bedpan; this must be free from antiseptics, and the stool must not be mixed with urine. It should be taken immediately to the laboratory so that it is examined whilst warm.

With a platinum loop, select a piece of blood-stained mucus, or, failing this, a small particle of faeces; emulsify it with a drop of warmed normal saline and apply a cover-slip.

The diagnosis of vegetative *Entamoeba histolytica* depends for practical purposes on the demonstration of actively motile amoebae, which contain red cells. The slide must be examined on a heated stage, or, if this is not available, it may be kept warm by applying halfpennies, pre-heated in a Bunsen flame, on each side of the cover-slip. Care must be taken not to overheat it. Motile amoebae are readily seen under the low power, and can be studied further under the  $\frac{1}{2}$ -objective. Iodine will kill the amoebae, and must not be used.

Cysts can be seen under the low power as small round refractile bodies. They are seen even better if the stool is emulsified in 1 per cent aqueous eosin, when, provided the stool is fresh, they show as white bodies against a pink background. The characteristic chromatoid bodies of *E. histolytica* are well shown by this method. Globules of oil or fat, which may be present in patients who have been given oil as an aperient, may resemble them, and, if numerous, may make any attempt at further examination useless. Oil droplets vary in size, are structureless, and their edges cannot be sharply focused. If cysts are present, iodine should be used for their further identification. Make a further preparation using 1 per cent Lugol's iodine. Find a suspected cyst under the low power. Apply the  $\frac{1}{2}$ -objective and centre the cyst in the middle of the field. Rack up the microscope tube, apply a small drop of oil to the cover-slip without moving it, and carefully lower the oil-immersion objective into the drop. The main differences between *E. histolytica* and *E. coli* and their cysts are shown in the table opposite.

	<i>Entamoeba histolytica</i> Vegetative Forms	<i>Entamoeba coli</i> Vegetative Forms
1 OCCURRENCE	Fairly abundant, when present, in amoebic dysenteric stools.	Never abundant. Occasionally seen in dysenteric stools.
2 SIZE	Variable, average 20 $\mu$ to 30 $\mu$ .	Less variable, on the average rather larger than <i>E. histolytica</i> .
3 MOTILITY	Active, large pseudopodia. These become blunter as the activity diminishes before death.	Sluggish. Blunt pseudopodia.
4 CYTOPLASM.	Homogeneous and 'ground-glass-like' (apart from food granules). The differentiation of ectoplasm and endoplasm may be clearly seen. Red blood-corpuscles often seen.	Appearance 'porcellanous'. Ectoplasm less plentiful, and line of demarcation between it and endoplasm inconspicuous. Endoplasm granular, abundant food vacuoles with usually bacterial inclusions. Red blood-corpuscles never present.
5 NUCLEUS	Fainter than in <i>E. coli</i> . Karyosome central with clear 'halo' round it. Periphery marked by ring-like layer of regular sized chromatin granules.	Distinct. Karyosome nearly always eccentric, 'halo' more definite. Ring-like layer of peripheral granules more pronounced and irregular in size and shape.

*Entamoeba histolytica*  
Cysts

When mature, four nuclei, with nuclear karyosomes central. Peripheral chromatin often semi-lunar. Size slightly smaller on average than *E. c.* cysts. Average size, 10  $\mu$  to 20  $\mu$ . Glycogen less abundant. Refractivity moderate. Rod-shaped 'chromidial bodies' usually seen in fresh specimen. Cyst-wall rather thinner.

*Entamoeba coli*  
Cysts

When mature, eight nuclei, with nuclear karyosomes usually eccentric. Size slightly larger than *E. h.* cysts. Vary from 10  $\mu$  to over 20  $\mu$ . Glycogen more abundant. Refractivity considerable. 'Chromidial bodies' not often present: thread-like or in bundles when seen. Cyst-wall rather thicker.

Besides *E. coli*, there are three other non-pathogenic amoebae, which must not be mistaken for *E. histolytica*. They are *Endolimax nana*, *Iodamoeba bütschlii* and *Dientamoeba fragilis*. In the diagnosis of the vegetative forms this mistake will be avoided if it is remembered that *E. histolytica* alone is actively motile and contains red cells. The cysts of *Iodamoeba bütschlii* (Plate XII, 8) contain a single small nucleus, a large compact mass of glycogen, but no chromatoid bodies.

*Giardia lamblia* is a flagellate protozoon, which inhabits the duodenum and may be found in the stools of patients with diarrhoea in both cystic and vegetative form (Plate XII, 9, 10). There is some doubt whether or not it is pathogenic. The fact that a course of meprazine will eradicate it, sometimes with relief of the diarrhoea, suggests that it is.

*Trichomonas hominis* (Plate XII, 11) is another flagellate protozoon, which may be seen in the stools in diarrhoea. It is probably non-pathogenic. Similar if not identical trichomonas may be found in the vagina in leucorrhoea and in the mouth in oral sepsis.

*Isospora belli* (*I. hominis*) (Plate XII, 12), one of a group of parasites which may produce coccidiosis in man, animals and birds, has occasionally been described as the cause of acute diarrhoea in man, particularly in the Eastern Mediterranean.

*Balantidium coli* (Plate XII, 13) is a large ciliate protozoon which is found in the intestine of pigs. It occasionally infects man, and may rarely cause severe diarrhoea or frank dysentery.

## 4 THE CIRCULATORY SYSTEM

### I ANATOMY

The *heart* lies obliquely in the thorax, being inclined from above downwards, forwards, and to the left (Plate XIII). Two thirds of it lie to the left of the middle line. The part which reaches highest in the thorax is the *left atrium*. It is usually opposite the 2nd interspace or lower border of the 2nd cartilage. The greater portion, however, of the *left atrium* lies posteriorly, and it forms the hindmost cavity of the heart.

The *right atrium* is the chamber that lies most to the right. It extends somewhat beyond the right margin of the sternum, and its border may be traced by a curved line joining the 3rd and 7th right sterno-costal articulations, and reaching about 1 in. to the right of the sternum.

The *right ventricle* occupies the greater portion of the front of the heart. Its inferior margin extends from the 7th right sterno-costal articulation to the apex, and it forms the lower border of the heart.

The *left ventricle* appears in front only as a narrow strip, scarcely  $\frac{1}{2}$  in. broad, and its outline completes that of the heart on the left, where its border forms a curved line, ascending from the apex to the lower margin of the 2nd left interspace at a point just internal to the parasternal line. The topographical anatomy of the valves of the heart and of the great vessels will be discussed in connection with auscultation (p. 77).

That portion of the anterior aspect of the chest which overlies the heart is known clinically as the *praecordium*. It is an area of indefinite size.

It is often necessary to define the exact situation of a point on the front of the thorax, and certain landmarks, some natural and some artificial, are commonly used for this purpose.

The ribs and interspaces on either side form convenient horizontal landmarks. In order to count them, feel for the ridge which marks the

junction of the manubrium with the body of the sternum, known as the angle of Louis, or sternal angle. When this has been found, run the finger outwards until it reaches the 2nd costal cartilage, which articulates with the sternum at this level. The space immediately above this is the 1st intercostal space. The spaces should then be counted downwards well away from the sternum, where they are more easily felt.

In order to define the distance of any given point from the mesial sagittal plane of the body, a series of vertical lines may be drawn on the chest. These are the *midclavicular line*, defined as the vertical line dropped from the centre of the clavicle, or, what amounts to the same thing, the line midway between the middle of the suprasternal notch and the tip of the acromion; and the *anterior, mid-, and posterior axillary lines*, descending respectively from the anterior border, the centre and the posterior border of the axilla.

Before proceeding to an examination of the heart, the student should feel the pulse and take the blood-pressure. The usual methods are inspection, palpation, percussion, and auscultation. In practice, inspection and palpation are often advantageously combined.

## II THE ARTERIAL PULSES

In cases of non-cardiovascular disease, it is usually sufficient to feel the radial pulse at the wrist. It is best felt with the tips of the fingers, the patient's forearm being pronated and the wrist slightly flexed. In cardiovascular disease, one should also feel the other radial, and the brachial, carotid, femoral, popliteal, posterior tibial and dorsalis pedis pulses, on both sides.

The following observations should be systematically made:

1. Rate of pulse.
2. Rhythm.
3. Character.
4. Volume.
5. Condition of vessel wall.
6. The presence or absence of delay of the femoral pulses compared with the carotids.

Estimates of the 'tension' of the pulse, that is of the blood-pressure within the vessel, by palpation, are quite unreliable. The blood-pressure should be determined with the sphygmomanometer. The terms 'good', 'bad', 'strong', and 'weak' in relation to the pulse lack precision and should be avoided.

1. The *rate* of the pulse is stated as so many beats a minute. It is counted, not when the fingers are first laid upon the pulse, but when any quickening due to nervousness of the patient has subsided and the pulse has resumed its normal rate. Count the beats for not less than half a minute. In cases of atrial fibrillation, the pulse rate counted at the wrist may not indicate the true rate of ventricular contractions. In all such cases, the rate of the heart beat should be counted by auscultation at the apex, and the difference between this rate and the pulse rate at the wrist should be recorded. This difference is referred to as the pulse deficit. The pulse-rate is increased during exercise, in fever and in thyrotoxicosis. It is also increased in paroxysmal atrial tachycardia, atrial fibrillation and atrial flutter (see Electrocardiography, p. 92). It is slowed somewhat in myxoedema, and in complete heart-block it beats at a steady 20–40 beats per minute.

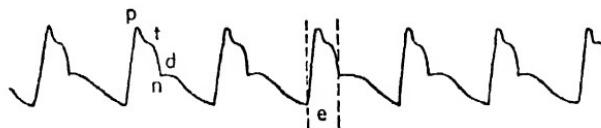


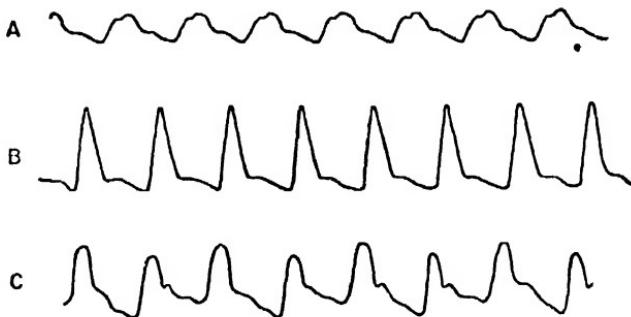
Fig. 4. Normal arterial pulse tracing.

*p* percussion waves; *t*, tidal wave; *n*, aortic notch; *d*, dicrotic wave; *e*, the period of ventricular systole (aortic valves open).

2. Decide next whether the *rhythm* is regular or irregular. If it is irregular, decide if it is completely irregular, whether the irregularity has a recurring pattern, or whether an otherwise regular rhythm is occasionally interrupted by some slight irregularity. The pulse of atrial fibrillation is completely irregular. The irregularity is usually obvious when the rate is rapid, but becomes less easy to recognize when the rate has been slowed by digitalis. If the rhythm has a recurring pattern, or there are occasional irregularities, these are likely to be due to extrasystoles. An extrasystole is a beat which occurs prematurely, is small and is followed by an unduly long pause. Disorders of rhythm are described more fully under Electrocardiography (p. 94).

3. Study the *character or form* of the individual pulse-wave. This is most satisfactorily done by palpating a large vessel, e.g. the carotid artery. It is not usually possible to detect the waves of the normal pulse, or slight variations from the normal, but in certain diseases the character of the pulse is detectably abnormal. The most important of these are as follows:

- (a) *Anacrotic pulse* (the term is a derivative of anadicrotic, meaning two up beats). This occurs in aortic stenosis, which gives rise to a slow ejection of blood from the left ventricle—the resulting pulse-wave has a slow upstroke, an anacrotic wave on the up-stroke, and the pulse is of small volume.
- (b) *Collapsing (water-hammer) pulse*. This is characterized by a rapid upstroke and descent of the pulse-wave. It occurs when there is an abnormal leak from the arterial system, for example aortic regurgitation, patent ductus, arterio-venous fistula, etc. It is best felt when the patient's arm is elevated and the wrist is grasped with the palm of the observer's hand against its palmar surface.



**Fig. 5. Arterial pulse tracing showing typical form of pulse-wave in (A) aortic stenosis, (B) aortic incompetence and (C) pulsus alternans.**

- (c) *Bisferiens pulse*. This is a combination of the anacrotic and collapsing pulses occurring in combined aortic stenosis and incompetence. The anacrotic wave is of the same height as the percussion wave, and both can be felt distinctly.
- (d) *Pulsus paradoxus*. The pulse becomes smaller, or even disappears at the end of inspiration, when the patient breathes deeply. This sign is found in pericardial effusion and in constrictive pericarditis.
- (e) *Pulsus alternans* (Fig. 5). When the ventricle beats strongly, then weakly, in successive beats of normal rhythm, alternation is present. In the radial tracing are seen alternate large and small beats, which are, however, *equidistant*. The condition is often

discovered when the systolic blood-pressure is taken, and the rate of sounds suddenly doubles as the pressure in the cuff falls.

When this condition is discovered, provided the heart-rate is moderate and no abnormal rhythm is present, it may be inferred that the heart-muscle is severely damaged.

4. Estimate the *volume* of the pulse beat, that is, the amplitude of movement of the vessel-wall during the passage of the pulse-wave. Provided that the arterial wall is normal, this is a measure of the pulse-pressure. If the vessel becomes rigid, however, commonly the pulse-pressure may widen whilst the pulse-volume on palpation may appear normal. The volume of the equivalent pulses on the two sides should then be compared.

5. The *condition of the vessel-wall*. Sufficient pressure should be exerted on the brachial artery to abolish pulsation in the radial vessel which should then be rolled beneath the fingers against the underlying bone. In young persons, the arteries cannot be felt or are soft. In older persons, they are more easily palpable. In arteriosclerosis they may feel hard like whipcord and may be tortuous.

6. Delay of the femoral compared with the carotid pulse is found in coarctation of the aorta.

The *typical pulse* of a healthy adult man should be described in the following terms. The rate is 70 per minute. The beats are regular in rhythm and equal in volume. The pulse is of normal volume and is not collapsing in character, the arterial wall is just palpable but is neither thickened nor tortuous.

### The Blood Pressure

The blood pressure is taken with a *sphygmomanometer*. The patient should be sitting or lying at ease. The manometer is placed so as to be at the same level as the observer's eye. All clothing should be removed from the arm. The cuff should be applied closely to the upper arm, with the lower border not less than 1 inch from the cubital fossa.

The radial pulse is palpated while the cuff is inflated to a pressure 30 mm. Hg. above the level at which radial pulsation can no longer be felt. The stethoscope is then placed lightly over the brachial artery. The pressure in the cuff is lowered, 5 mm. Hg. at a time, until the first sound is heard, which is the systolic pressure. Continue to lower the pressure in the cuff until the sounds become suddenly faint or inaudible: this is the diastolic pressure.

**Precautions.** Arterial pressure shows temporary variations with change

of posture, after meals, on exertion, and notably on excitement. Hence it should be observed only after the patient has been reassured, and when he is quietly resting, free from excitement and with the arm relaxed. In nervous patients the first reading is often too high and should be rejected; a second reading will more closely represent the true pressure. The pulse rate at the time should be noted, for blood-pressure varies to some extent with the rate of the heart. It is essential to work as quickly as is compatible with accuracy, for compression of a limb itself induces a rise in blood-pressure. To reduce this source of error when successive estimations are to be made, the air-pressure in the armlet should always be allowed to fall to zero as soon as each reading has been taken. It is important to take the blood-pressure of patients taking hypotensive drugs in both the recumbent and the standing positions.

Occasionally the sounds disappear at a point below 200 mm. for a period and then reappear, finally disappearing at the point of diastolic pressure. Thus the sounds may first appear when the mercury falls to 210 (systolic pressure), disappear from 180 to 160 (silent gap), reappear and finally disappear at 120 (diastolic pressure). This phenomenon of a *silent gap* is found in certain patients with hypertension; its significance is unknown, but its occurrence makes it important that the armlet pressure should always be well raised at the beginning of an estimation of blood-pressure.

It is important to check that the width of the cuff is correct. For an adult, the standard cuff width is 12·5 cm. If a narrower cuff is used, the recorded pressures will be falsely high.

For children, there is a variety of cuffs of different widths. One should select the size which covers most of the upper arm but leaves a gap of  $\frac{1}{2}$  in. below the axilla and above the antecubital fossa.

Occasionally it becomes necessary to compare the systolic blood-pressure in the arm with that in the leg. The patient lies face downwards and a seven-inch cuff is applied above the knee and auscultation carried out over the popliteal artery. In coarctation of the aorta, the blood-pressure in the legs is lower than in the arms.

**Normal blood-pressure.** The average systolic pressure in healthy adults is 100–140 mm. Hg., the average diastolic pressure, 60–90 mm. In children it approximates to the lower figure in each case, and in the elderly it reaches or even exceeds the higher figure. The difference between the systolic and the diastolic pressures—the pulse-pressure—is 30–60 mm.

**Abnormal blood-pressure.** A high systolic blood-pressure with a normal diastolic pressure (systolic hypertension) is frequently encountered in the elderly, and is a function of inelasticity of the arteries (arteriosclerosis).

A raised diastolic pressure is of much greater significance, and should lead to a search for a primary cause such as renal disease, Cushing's syndrome, phaeochromocytoma, etc. If these causes are excluded, a diagnosis of idiopathic (or essential) hypertension is made.

### III INSPECTION AND PALPATION

The patient should be examined in a good light, sitting up at an angle of 45° if this is possible. The observer should first note the presence or absence of dyspnoea and cyanosis and the shape of the chest and praecordium. He should then turn his attention to:

1. The neck veins.
2. Veins on the chest wall.
3. The cardiac impulse.
4. Other pulsations.
5. Thrills.

#### 1 The Neck Veins

The neck veins communicate directly with the right atrium. Changes in the mean pressure within them, together with the pulsations that occur with each cardiac cycle, give direct information, therefore, about mean pressure in the right atrium and about pressure-changes during the cardiac cycle.

The neck veins should be examined with the patient in a good light, and reclining at an angle of about 45°. The veins normally show slight pulsation, and three small waves can be distinguished in each cardiac cycle. There is, however, a mean level, and the perpendicular height of this level above the right atrium indicates the mean hydrostatic pressure within the right atrium. In health this level is the same as that of the manubrium sterni, whatever the position of the subject. The manubrium is therefore a convenient reference point for measuring or estimating the right atrial pressure. This means that in a healthy person reclining at an angle of 45°, the mean level will be invisible, because it is below the clavicle, but some slight pulsation may appear above the clavicle.

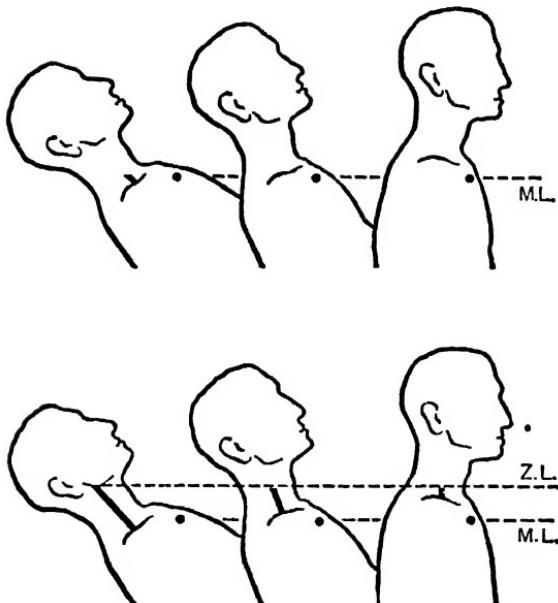


Fig. 6.

In health vertical height of column of blood in jugular veins is about level with manubrium sterni (ML), whatever the position of the patient. In heart failure, when the right atrial pressure is increased, the vertical height of this column is increased and is above the manubrial level (ML), whatever the position of the patient. (Modified from Lewis's *Diseases of the Heart*, 2nd ed., London.)

Arterial pulsation may also be visible in the neck, and venous pulsation has to be distinguished from it. The venous pulse has a definite upper level, though it may be necessary to sit the patient up higher or lay him lower to find it. This level falls during inspiration when blood is drawn into the heart. Firm but gentle pressure on the abdomen will raise the level by increasing the venous pressure generally. Venous pulsation is usually more sinuous and less sharp than arterial pulsation. Finally it is impalpable; or even when grossly abnormal, e.g. in tricuspid incompetence, it is only just palpable while arterial pulsation is easily palpable and thrusting in character.

A raised venous pressure is usually indicative of right heart-failure. Occasionally it is due to obstruction of the superior vena cava, in which case the normal pulsations of the venous pulse are absent. A slight rise in venous pressure also occurs with an increase in the circulating blood-volume, as in pregnancy and acute nephritis.

No cardiac case has been properly examined till the level of the venous pressure in the neck has been determined. This level is usually seen quite easily by the methods described, but occasionally difficulties occur. The mean venous pressure may be so high, for instance, that the pulsation is obscured behind the jaw when the patient is semi-recumbent, and visible only high in the neck when he sits upright. Further, too much reliance should not be placed on the external jugular veins, as these may only be superficial for part of their course. Internal jugular pulsation is a more reliable guide to the venous pressure.

The early studies of the venous pulse were made with the polygraph, which consists of a tambour recording on paper which is moved by a clockwork mechanism. A simultaneous trace of the arterial pulse was used for timing purposes. The use of this technique at the beginning of this century really marks the advent of modern cardiology.

The venous pulse has three positive waves *a*, *c*, and *v* and two negative waves or descents *x* and *y*. The *a* wave is due to atrial contraction. This is followed by the *x* descent, which is interrupted by a small *c* wave (which is

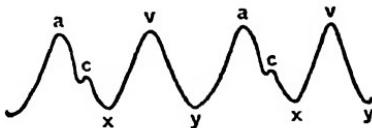


Fig. 7. The venous pulse.

rarely visible on inspection of the neck veins). The *c* wave coincides with the onset of ventricular systole and results from tricuspid valve closure. The *v* wave indicates a rise in pressure as venous return continues while the tricuspid valve is closed. When the tricuspid valve opens, blood enters the right ventricle rapidly and there is consequently a lowering of the right atrial pressure—the *y* descent.

Usually it will be found that the venous pressure is normal, and that the waves of the venous pulse are small and of approximately equal amplitude. If, however, one of the venous waves is increased, and producing a pulsation of 3 cm. or more, it should be identified. If the thumb is placed on the carotid artery at the angle of the jaw, a venous wave seen just before the arterial pulse (presystolic) will be an *a*, and one just after a *v*. An abnormally large *a* wave tends to be 'flicking' in character, a *v* slower and more expansile. It is worth while recording the approximate vertical height of the peak of the wave above the manubrium, for this is its pressure in centimetres of water (or very approximately in millimetres Hg. since 1 mm. Hg. is

approximately equal to 1·3 cm. of water). The *a* wave is prominent when the resistance of the right ventricle to filling is increased, as in tricuspid stenosis, or from hypertrophy of the right ventricle due to increased pressure work, in pulmonary stenosis or pulmonary hypertension. In tricuspid incompetence, the *v* wave is replaced by a large wave due to regurgitation of blood into the right atrium during right ventricular systole. This regurgitant flow of blood may well exceed the forward flow through the heart, and explains why the venous wave in tricuspid incompetence has such a surging expansile character.

Obviously *a* waves will disappear when there is no active atrial contraction in atrial fibrillation. In complete heart-block (complete atrio-ventricular dissociation) regular *a* waves can be seen in the neck while carotid pulsation occurs at a slower independent rate. From time to time atrial contraction occurs when the atrio-ventricular valves are closed during ventricular systole. All the force of right atrial contraction is transmitted into the veins, to give a 'cannon wave'. If the arterial pulse is abnormally slow, the neck veins should be carefully examined for more rapid, regular *a* waves and cannon waves. Cannon waves are also seen in nodal rhythm when the atrium and ventricle are activated simultaneously, and the atrium contracts on closed atrio-ventricular valves.

## 2 Veins on the Chest Wall

The veins of the thoracic wall may be unduly conspicuous. This occurs (*a*) when the patient's skin is unusually transparent; (*b*) when an intrathoracic growth or aneurysm obstructs the return of blood to the heart; (*c*) when, in consequence of portal obstruction or obstruction of the inferior vena cava, the blood returning from the abdominal viscera or lower limbs is forced to find its way through collateral channels.

## 3 The Cardiac Impulse

It is customary to locate the 'apex beat', which is the lowest and outermost point of definite cardiac pulsation, and to locate its position in terms of the particular intercostal space and distance from the mid-line in which it is felt. The normal position of the 'apex beat' is 3½ in. from the mid-line, or half an inch internal to the mid-clavicular line in the fifth intercostal space.

The position of the 'apex beat' is a valuable physical sign, if its limitations are understood.

First, it must be felt with the patient sitting up straight or lying quite flat. The 'apex beat' of a normal person may be felt in the anterior axillary line, if the person is lying on his left side.

Secondly, it must be realized that the commonest cause of displacement of the 'apex beat' is a deformity of the thoracic cage, usually scoliosis.

A real displacement of the 'apex beat' can occur in three types of case.

The cause may be (a) congenital, when the heart is reversed so that the apex lies to the right (dextrocardia); (b) extrinsic, when the heart is displaced by disease of the surrounding viscera, which 'push' or 'pull' it from its usual site. Instances of 'pushing' are found in pleural effusion and pneumothorax, and of 'pulling' in pulmonary fibrosis and collapse of the lung: (c) disease of the heart, when displacement of the 'apex beat' may indicate enlargement, particularly of the left ventricle. Of much greater importance in this connection than the exact position of the apex beat is palpation over the left ventricle, the right ventricle, and the pulmonary artery to determine the character of the cardiac impulse, from which information can be gained about the function of the ventricles.

The *left ventricle* normally produces the 'apex beat'. When it is hypertrophied, the beat becomes more forceful and may extend outwards towards the axilla.

The *right ventricle*, when hypertrophied, can be felt by placing the hand firmly over the praecordium just lateral to the sternum, when a definite 'lift' will be detected.

The *pulmonary artery* can be palpated (when enlarged) in the second left intercostal space on expiration.

If an hypertrophied ventricle is present, an attempt should be made to differentiate between that resulting from obstruction to outflow (systolic overload) and that associated with excess filling of the ventricle (diastolic overload). Systolic overload, due to aortic stenosis or systemic hypertension, causes a forceful sustained heave. On the other hand, the excessive diastolic expansion, occurring in aortic and mitral regurgitation, causes diastolic overload, and is followed by easy and rapid ejection of blood from the ventricles, producing an equally forceful but less sustained impulse.

#### 4 Other Pulsations

In addition to the pulsations already described, movements should be looked for at the root of the neck, the front of the chest, and the epigastrium.

*In the suprasternal notch* the pulsation is usually systolic in time, and when well marked may be an indication of raised aortic arch in

hypertension or coarctation of the aorta; or of aneurysm of the arch of the aorta.

In the neck various pulsations may be observed. These may be either arterial or venous. The latter has already been described. The carotids pulsate visibly on exertion; from mental excitement; in diseases which cause overaction of the heart, such as thyrotoxicosis; in cases of aortic incompetence, hypertension and aneurysm of the aorta. In hypertension, especially in women, and in association with a high aortic arch, the right carotid sometimes shows abnormal pulsation due to kinking, which must not be mistaken for aneurysm.

In the thorax, a rare source of pulsation in unusual parts is aneurysm of the aorta. Such aneurysmal pulsations always manifest themselves at first above the level of the 4th rib, though at a later period they may affect a considerable portion of the chest-wall. The position of the impulse varies according to the part of the aorta which is diseased. If the ascending aorta is affected, the pulsation is chiefly to the right of the sternum, whilst the transverse aorta gives rise to less distinct pulsation under the manubrium sterni, and the descending aorta still more to the left. The time of this pulsation is systolic, following immediately on the apex-beat, and it may be observed to be expansile in character.

In coarctation of the aorta a collateral arterial circulation develops, and pulsation may be detected in superficial arteries in the chest-wall. This may best be seen over the back with the patient bending forward to touch his toes (Plate VIII).

Pulsation in the epigastrium is most commonly due to nervousness or excitement in a thin person. Less commonly it is due to a hypertrophied right ventricle, or to transmission of the aortic pulsation by a tumour, such as a carcinoma of the stomach. Occasionally it is due to a disensible pulsation of the liver in heart failure with tricuspid incompetence; and very rarely to an aneurysm of the abdominal aorta, which may be palpable as an expansile swelling.

## 5 Thrills

Any sound or murmur which is loud will be palpable. A palpable murmur is called a thrill and transmits to the hand a sensation like the purring of a cat. The character, timing, and variation with respiration of thrills are the same as those of the corresponding murmurs and are discussed with murmurs. An abnormally loud sound is felt as a shock. A third sound or atrial sound is often easier to feel than

to hear. The loud first sound of mitral stenosis is often palpable and the sounds of aortic and pulmonary valve closure are often palpable in systemic and pulmonary hypertension respectively.

#### IV PERCUSSION

Percussion of the heart, as a means of detecting slight changes in the size of that organ or of its chambers, has been shown to give false results. For these purposes X-ray methods are now used (p. 106). The student must, however, learn to percuss the cardiac dullness in order to detect the presence of large pericardial effusions and aortic aneurysms, and the absence of the cardiac dullness in hypertrophic emphysema.

By percussing in the 4th interspace from the left lung towards the heart it is possible to define the left border more or less precisely. It is found about half an inch internal to the mid-clavicular line. The right border of the heart is just to the right of the sternum at the level of the fourth rib. It is difficult to define, since the sternum acts as a sounding-board.

In *aortic aneurysm* a dull area can often be mapped out. Below, it is continuous with that of the heart; above, it bulges outwards to the right of the sternum at the level of the 2nd interspace and adjacent ribs; and the sound produced by percussion of the manubrium sterni is also rendered less resonant, or even absolutely dull when the aneurysm is large.

In *pericarditis with effusion* the dullness varies with the amount of fluid which is present, but in well-marked cases is pear-shaped, with the broader end downwards and the upper end higher and broader than the ordinary upper limit of dullness.

In *hypertrophic emphysema* the area of cardiac dullness is reduced.

#### V AUSCULTATION

To become skilled in auscultation of the heart requires a great deal of practice. Eventually one becomes attuned to the various sounds and murmurs, and learns to focus attention on one portion of the cardiac cycle at a time. It is wise to palpate the carotid artery while auscultating, to avoid the common error of mistaking systole for diastole and vice versa.

*The stethoscope.* The most important features of the stethoscope

are that extraneous noises must be excluded as far as possible and that the conduction of sound from the chest-pieces should be as complete as possible. In order to achieve these, the ear-pieces should be well-fitting and there should be no leaks elsewhere. The tubes should be of small diameter, relatively short, and with polished inner surface to reduce loss of conduction by friction. Plastic tubing is much more efficient in this capacity than rubber.

### Auscultatory areas

- It is customary to listen first in the following areas:
- *The mitral area*—which corresponds to the apex beat.
  - *The tricuspid area*—which lies just to the left of the lower end of the sternum.
  - *The aortic area*—which is to the right of the sternum in the second intercostal space.
  - *The pulmonary area*—which is to the left of the sternum in the second intercostal space.

It must be appreciated (i) that auscultation must not be confined to these areas, and (ii) that noises heard in a particular area do not necessarily come from that particular valve; for example murmurs originating at the aortic valve are frequently best heard at the mitral area. There is usually, however, good correlation.

*The events of the cardiac cycle* are illustrated in Plate XX. At the onset of ventricular systole, the mitral and tricuspid valves close almost simultaneously to give the *first heart sound*. The opening of the aorta and pulmonary valves occurs next and is inaudible. The closure of the aortic and pulmonary valves gives rise to the two components of the *second sound*. It will be seen that because of the lower pressure in the right ventricle compared with the left, closure of the pulmonary valve follows that of the aortic valve. After a brief period the mitral and tricuspid valves open inaudibly in the normal heart.

### Deviations from the normal in disease

- In disease the following deviations from the normal may occur:
1. The sounds may have a different intensity, both absolutely and relatively to each other, from that possessed in health.
  2. The sounds may be split.
  3. A triple rhythm may be present.
  4. Adventitious sounds may be heard, either replacing or occurring along with the heart sounds.

## 1 Alterations in Intensity

In patients with thick chest-walls, and in those with a serious degree of emphysema, the heart sounds may scarcely be audible, though there is no heart disease. Conversely, in the presence of serious heart disease the sounds may appear quite normal. Thus alterations in the intensity of the heart sounds are significant only when considered in relation to all the other features of the case. The heart sounds are distant or inaudible in pericardial effusion. Accentuation of the first sound is often present in mitral stenosis and in tachycardia from any cause.

An absolute accentuation of the sound of aortic or pulmonary valve closure is found when there is systemic or pulmonary hypertension.

## 2 Splitting

The mitral valve closes slightly before the tricuspid valve, and this can give rise to splitting of the first sound. This splitting is difficult to detect by auscultation and even by phonocardiography, because both components are very low-pitched and merge one into the other. When it is heard, splitting of the first sound is not a sign of heart disease and is of importance only because its two components may be confused with an atrial sound and a first sound, or with a first sound and an ejection click.

Splitting of the second sound is much easier to hear, because the aortic and pulmonary valve closure sounds ( $A_2$  and  $P_2$ ) have high-pitched harmonics, and the two sounds can be separated, especially when the diaphragm is used for auscultation. Aortic valve closure ( $A_2$ ) is audible in all areas. Pulmonary valve closure ( $P_2$ ) is audible only in the pulmonary area and for a short distance down the left sternal edge, unless its intensity is greatly increased due to pulmonary hypertension. It follows that splitting of the second sound is usually heard only at and close to the pulmonary area. Splitting is most easily heard in children, and may not be audible in older adults, especially men, when muscle noise, a thick chest wall, and emphysema make  $P_2$  inaudible. Normally  $P_2$  always follows  $A_2$ , and the splitting is widest during inspiration and narrowest in expiration, when the two components usually merge to give a single sound. Splitting of 0·06

sec. during inspiration and 0.02 sec. (a very close split or single sound) in expiration would be average for a child or young adult.

The mechanism of splitting of the second sound is as follows. During inspiration blood is drawn into the thorax, there is a relative rise in right atrial pressure, and the right ventricular stroke-volume increases. The duration of right ventricular systole measured from the first sound to  $P_2$  is increased, and  $P_2$  is therefore slightly delayed. Conversely the left ventricular stroke-volume falls during inspiration, because the greater negative pressure within the thorax enlarges the capacity of the left atrium and pulmonary veins and reduces left atrial pressure and hence left ventricular filling and stroke-volume. Thus left ventricular systole is shortened and  $A_2$  is earlier. During inspiration, then,  $A_2$  occurs earlier and  $P_2$  later, so that splitting of the second sound widens. During expiration the changes are exactly opposite and the splitting narrows. Movement of  $P_2$  is considerably greater than that of  $A_2$ . This concept of respiratory variation in right and left ventricular stroke-volume is of the greatest importance.

### 3 Triple Rhythm

Phonocardiography shows that in addition to the two heart sounds generally recognized, a third sound and an atrial sound are often present. When either of these additional sounds are prominent and audible, they give a cadence of sounds known as triple rhythm.

The third sound follows the aortic component of the second sound by about 0.15 sec. It is usually best heard in the mitral area and is lower pitched than the second sound, which it follows. Triple rhythm from a third sound is common in young persons, and can occur in health up to the age of 40. It is an important sign in heart failure from any cause, and may be heard shortly after cardiac infarction. (The third sound occurs at a time when the ventricle is filling very rapidly, and in disease when the distensibility of the ventricular muscle is likely to be altered. For this reason, it used to be thought to be a 'filling-sound', but recently there has been evidence that it originates in the atrio-ventricular valves. Theoretically, a third sound could arise on either side of the heart, but in fact it nearly always arises in the left heart.)

The atrial sound is a low-pitched sound occurring before the first sound. It is not heard in health. Triple rhythm from an atrial sound can originate in the right heart from pulmonary stenosis or pulmonary hypertension, and in the left heart from systemic hypertension. It is not a sign of failure but does indicate that the heart is under strain. Like the third sound, it used to be thought to be a 'filling sound', but now a valvular origin is suspected.

When the heart-rate is rapid (100 or more), diastole tends to be relatively shortened compared with systole, and a third sound may come to

overlie an atrial sound, giving rise to a 'summation gallop'. Usually, when the heart slows, a loud third or atrial sound is audible.

#### 4 Murmurs

Murmurs have a blowing or musical quality. They are due to turbulence in the blood-flow at or near a valve or an abnormal communication within the heart. Not all murmurs are due to organic damage in the heart; they may be due to such causes as an abnormally rapid flow of blood through a normal valve. Such murmurs are called flow murmurs. In examining a murmur the following points must be noted:

- (a) **Its time of occurrence.** Murmurs may be (i) systolic, (ii) diastolic or (iii) continuous throughout systole and diastole. *Systolic murmurs* are either *pan-systolic*, as in mitral and tricuspid regurgitation and ventricular septal defect, or *effection*, when they arise either at the pulmonary or aortic valves. *Diastolic murmurs* are either *immediate* (or early), starting at the second heart-sound and occurring as a result of aortic or pulmonary regurgitation; or *delayed* (or mid), when there is a short gap after the second heart-sound and the beginning of the murmur. These murmurs arise at the mitral or tricuspid valves.
- (b) **The behaviour of the murmur during respiration.** The stroke output of the right heart increases during inspiration, while that of the left heart is reduced. It follows that a murmur originating on the right side of the heart will become louder during inspiration.) Because the chest wall tends to be carried away from the heart during inspiration, and the lung tends to cover the heart, most cardiac events are not so well heard during inspiration. Any murmur, therefore, which increases on inspiration can be attributed to the right heart; and any murmur which increases on expiration can be attributed to the left heart.
- (c) **Its point of maximum intensity and direction of selective propagation.** The maximum loudness of a murmur which has been produced at a given valve usually occurs at the point where the valve-sound would be best heard in health. To this rule, however, there are some exceptions.

Murmurs are not equally well heard at all points on the chest-wall which are equidistant from the point of their

greatest intensity, but each is much more distinctly audible in some directions than in others; i.e. such murmurs have a direction of selective propagation.

- (d) **The character of a murmur.** This also helps to determine its origin. Obstructive murmurs, from obstruction to the onward flow of blood through a narrowed valve, are usually rough; regurgitant murmurs, from leakage backwards through a closed but incompetent valve, are softer and blowing. The loudness of a murmur has no relation to its importance. A very loud murmur may be less significant than one so soft as to be nearly inaudible.

It has already been stated that murmurs are due to turbulence in the flow of blood. The most important cause of turbulence within the heart is the rapid flow of blood through a relatively small orifice, and this means that there must be a considerable difference in pressure on the two sides of the orifice leading to a sufficient gradient. In the normal adult heart, the pressure gradients across open valves are very small, because the valve orifices are fairly large in relation to the flow of blood through them. There are therefore no murmurs. If, however, a valve orifice should be narrowed by disease, a sufficient pressure-gradient may build up at certain times during the cardiac cycle to produce a murmur.

In the next paragraphs the haemodynamics in some common forms of valvular disease are presented, in order to show how murmurs are produced and why they arise when they do. Certain abnormal valve-sounds which are associated with the opening and closing of abnormal valves will also be mentioned, and a note of the associated clinical findings will be included.

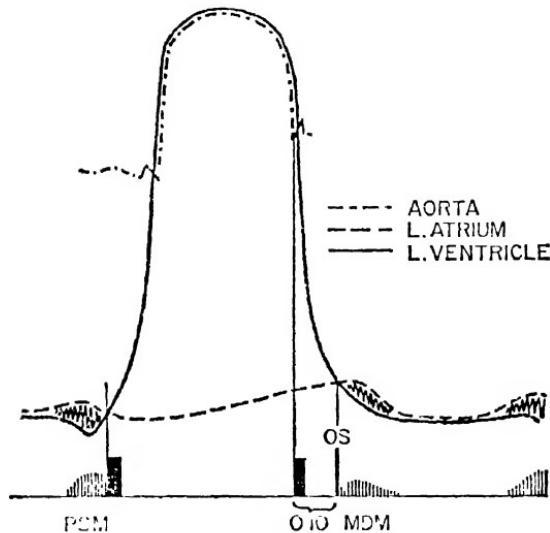
## 1 Mitral Valve Disease

### Mitral Stenosis

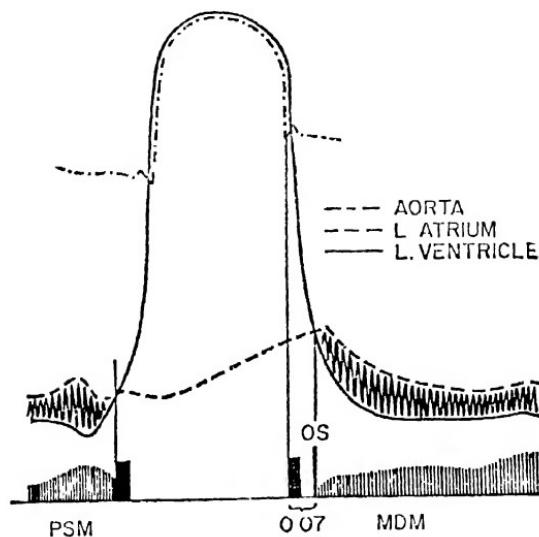
Narrowing of the mitral valve orifice, mitral stenosis, is due to the fusion of the two valve cusps along their margins, extending from the valve ring at the periphery in towards the centre. The normal valve closed by the apposition of two mobile cusps over a length of 3·5 cm. becomes a fibrotic diaphragm with a small central orifice closed by the apposition of 1 cm. or less of cusp tissue.

The haemodynamics in a mild and a severe case of mitral stenosis are shown diagrammatically in Figs. 8, 9.

*Loud first heart-sound.* At the onset of systole, the left ventricular



**Fig. 8.** Diagram showing the relation of murmurs to haemodynamics in mild mitral stenosis.



**Fig. 9.** Diagram showing the relation of murmurs to haemodynamics in severe mitral stenosis.

pressure begins to rise, and the mitral valve closes when this exceeds the left atrial pressure. In mitral stenosis, the left atrial pressure is raised, and the left ventricle is contracting vigorously when this occurs. The mitral valve therefore closes very rapidly and a loud first heart-sound results.

*The opening snap* of the mitral valve is a loud sound heard just after the second heart-sound. It is a consequence of the high left-atrial pressure, which forces the mitral valve open rapidly when the ventricle relaxes. If the mitral valve is rigid and calcified, it follows that the first heart-sound will not be accentuated and the opening snap will not be present.

*The mitral diastolic murmur* is a low-pitched rumbling murmur heard at the apex; it is due to blood passing through the narrowed mitral valve when there is a pressure-gradient between the left atrium and left ventricle. (In the normal heart no such gradient exists and the pressures equalize rapidly when the mitral valve opens.)

*The presystolic murmur* results from atrial systole, and therefore immediately precedes the first heart-sound. It is due to an increase in flow across the narrowed mitral valve during atrial contraction. If atrial fibrillation is present, in which there is no effective contraction of the atria, the presystolic murmur is obviously not present.

*Mild mitral stenosis*, is associated with a slightly raised left-atrial pressure. Mitral valve opening occurs at a normal time, so that the opening snap is relatively late and the diastolic murmur is short.

*Severe mitral stenosis* causes a considerably raised left-atrial pressure. The opening snap occurs early and the diastolic murmur is long.

The auscultatory signs in mitral valve disease may be sharply localized at or near the apex. It is always worth-while listening with the patient lying on his left side, as this throws the heart up against the chest wall and apical murmurs are better heard. In cases of mild mitral stenosis the patient should be examined after exercise, for this increases the cardiac output and hence accentuates the pressure gradients and the murmurs.

### **Mitral Regurgitation**

In mitral regurgitation the cusps of the mitral valve fail to close completely during ventricular systole, which results in a jet of blood being forced back into the left atrium throughout systole, leading to a pan-systolic murmur.

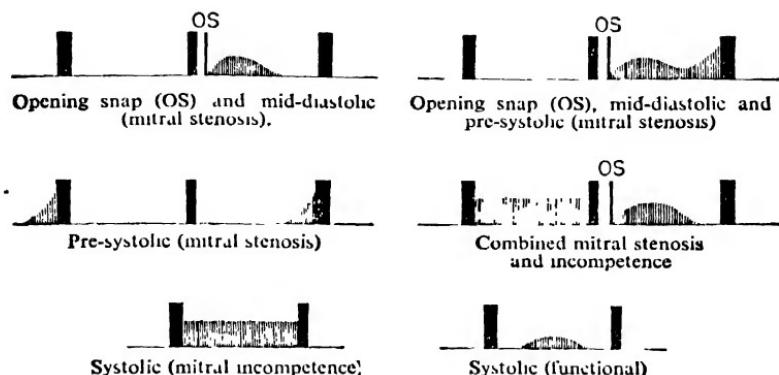


Fig. 10. Mitral murmurs.

*The mitral pan-systolic murmur* is due to blood leaking back through the mitral valve in ventricular systole. It starts therefore when the mitral valve normally closes, i.e. with the first heart-sound, and continues throughout systole. It is best heard at the mitral area, and radiates into the axilla and increases in intensity with expiration.

*The soft first heart-sound.* The bulk of the first heart-sound is due to mitral valve closure, and if this closure is imperfect, the resulting heart-sound will be softer than normal.

## 2 Aortic Valve Disease

### Aortic Stenosis

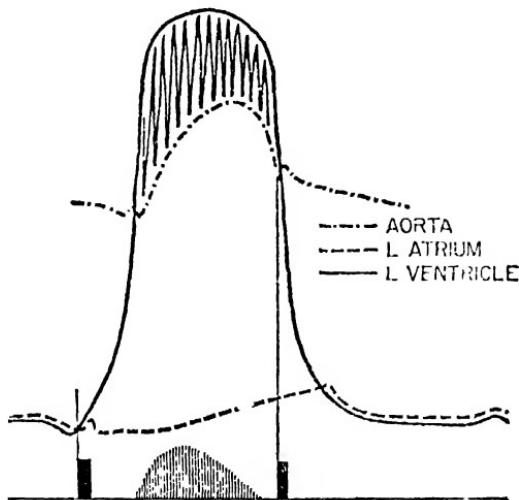
The haemodynamics in aortic stenosis are shown diagrammatically in Fig. 11. There is a considerable pressure gradient between the left ventricle and aorta, and this gradient is greatest in the middle of systole, and is relatively small early and late in systole. The murmur of aortic stenosis is therefore mid-systolic. All murmurs due to the ejection of blood through abnormal semilunar valves, or to abnormally high blood flow through normal semilunar valves, have this same pattern and are called 'ejection' systolic murmurs.

**The aortic ejection murmur.** This murmur is heard in the aortic area and radiates into the neck although it is frequently well heard at the mitral area. It increases on expiration.

*The aortic ejection click* is due to opening of the aortic valve in

aortic stenosis. It is therefore heard just after the first heart-sound at the beginning of the ejection murmur.

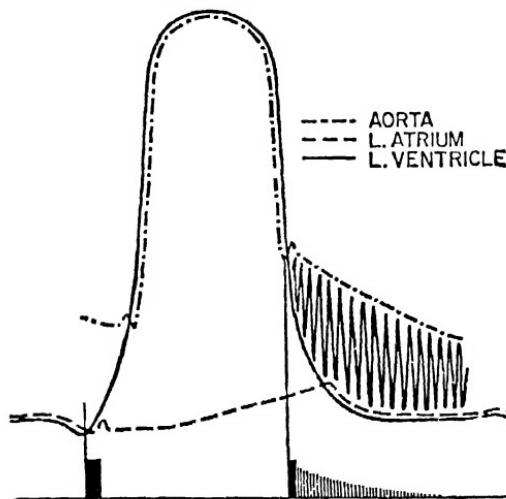
*Delayed and soft closure of the aortic valve* are much less easily detectable signs of aortic stenosis. The aortic valve closure is delayed because it takes longer for the left ventricle to expel its blood through the narrowed valve. Pulmonary valve closure may then precede the aortic closure. This is known as 'reversed splitting' of the second sounds. Aortic closure will also be soft simply because the mobility of the semilunar cusps is reduced and they do not snap together with the usual force.



**Fig. 11. Diagram showing the relation of murmurs to haemodynamics in aortic stenosis.**

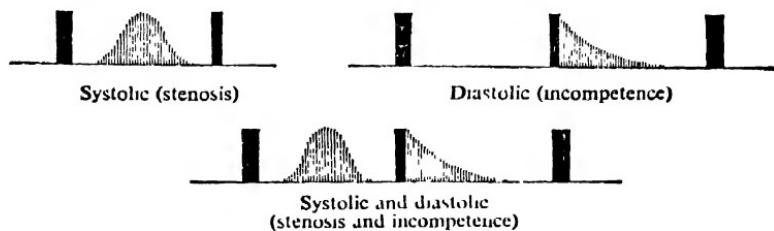
### Aortic Regurgitation

The aortic leak is surprisingly large, and in severe incompetence may equal the forward flow of blood into the circulation. That is to say that double the normal stroke-output may be ejected through the aortic valve into the aorta during systole, and this may give rise to an aortic ejection-murmur. Thus an aortic ejection-murmur in a patient with aortic incompetence does not necessarily indicate a concomitant stenosis.



**Fig. 12.** Diagram showing the relation of murmurs to haemodynamics in aortic incompetence.

*The early or immediate diastolic murmur* starts with the second heart-sound and continues for a variable time in diastole. It is a high-pitched murmur, usually soft, and often requires intent auscultation for its recognition. It is best heard down the left sternal edge when the patient sits forward and breathes out, and the examiner listens with the diaphragm of his stethoscope.



**Fig. 13.** Aortic murmurs.

### 3 Tricuspid Valve Disease

Organic tricuspid valve disease is almost always of rheumatic aetiology, and is almost always accompanied by rheumatic mitra

and aortic valve disease. The diagnosis can often be made from inspection of the neck veins (p. 71). The murmurs of tricuspid stenosis and of tricuspid incompetence have the same timing and character as the corresponding mitral lesions, but are best heard at or near the lower end of the sternum and are louder in inspiration. Tricuspid opening snaps are rare.

Functional tricuspid incompetence is often found with congestive failure in atrial fibrillation from any cause. It is due to dilatation of the tricuspid valve ring, consequent on dilatation of the right ventricle, and the characteristic changes in the venous pulse disappear or diminish when the heart failure is controlled.

#### 4 Congenital Heart Disease

##### Pulmonary Stenosis

In order to maintain the cardiac output in the presence of pulmonary stenosis the right ventricular pressure has to rise considerably and exceeds the level of systemic pressure in severe cases. In mild pulmonary stenosis the only abnormal signs are found on auscultation. The ejection systolic murmur (and thrill if present) is most prominent in inspiration and best heard in the pulmonary area. It is usually preceded by an ejection click which, surprisingly, is loudest in expiration. The second sound behaves normally on inspiration, but the width of splitting may be slightly wider than normal because of a delay in pulmonary valve closure. If the stenosis is severe, the closure of the pulmonary valve becomes soft or inaudible.

##### Atrial Septal Defect

Blood flows through the defect from left atrium to right, since the normal pressure-relationships are maintained. The blood-flow through the defect is often equal to twice the flow entering the left ventricle (i.e. the systemic blood-flow). The flow of blood entering the right atrium will then be the systemic flow plus the flow from the left atrium through the atrial septal defect—i.e. three times the systemic flow. This torrential flow passes from the right atrium to the right ventricle, pulmonary artery, lung vessels and so back to the left atrium.

Because the defect is very large and the pressure-gradient very small (about 1 mm. Hg.), there is little turbulence and no murmur from blood flowing through the defect. The characteristic auscultatory sign is the relatively wide splitting of the second heart-sound, uninflu-

enced by respiration, as has been discussed. The torrential blood-flow through a normal pulmonary valve produces a functional ejection pulmonary systolic murmur which is moderately loud. A functional tricuspid flow murmur is often present. It is mid-diastolic, soft, and best heard at the lower end of the sternum during deep inspiration.

### Ventricular Septal Defect

Ventricular septal defects are rarely as large as atrial septal defects and commonly range from the size of a sixpence downwards. The right ventricular systolic pressure is normally about one-fifth of that in the left ventricle, so that there is a pressure-gradient from left ventricle to right throughout systole.

In mild cases (*maladie de Roger*) where the defect has the cross-sectional area of a pencil or smaller, the only abnormal sign is a pan-systolic murmur, loudest in expiration, usually accompanied by a thrill, and best heard at the lower end of the sternum.

In more severe cases there is evidence of increased blood-flow through the left heart and blood-vessels of the lung. In addition to the pansystolic murmur and thrill there may be a soft short mid-diastolic murmur due to the rapid flow of blood through a normal mitral valve. Splitting of the second sound is normal, though  $A_2$  may be difficult to hear, as it tends to be obscured in the loud pan-systolic murmur.

### Patent Ductus Arteriosus

The communication has the same size range as in ventricular septal defect, but its length also tends to limit flow. The pressure gradient from aorta to pulmonary artery is present throughout the cardiac cycle and is greatest towards the end of systole. The increased flow affects the left heart and pulmonary circulation exclusively.

Again, if the communication is small, a murmur is the only abnormal sign. It is best heard at the pulmonary area in expiration, and is called continuous. The accentuation of the continuous murmur about the time of the second sound gives it a particular character, and it is sometimes described as a machinery murmur. In addition to the continuous murmur, a short soft functional mitral diastolic murmur may be present.

A continuous murmur is not specific for patent ductus arteriosus, but can be produced by the rapid flow of blood through any narrowed vessel. Patent ductus arteriosus is the commonest of several such possible conditions within the thorax. Particular care should always be taken not to confuse it with a venous hum.

**Fallot's Tetralogy**

The tetralogy consists of pulmonary stenosis, ventricular septal defect, right ventricular enlargement, and overriding of the aorta, i.e. the aorta arises astride the ventricular septal defect, so that blood enters it from the right ventricle and the left ventricle. The essential features are pulmonary stenosis of at least moderate severity, and a large ventricular septal defect, so that the pressure in the two ventricles is equalized. The right ventricular output is ejected partly into the pulmonary artery through the pulmonary stenosis, and partly into the aorta through the ventricular septal defect. The admixture of deoxygenated blood with the left ventricular output makes the patient cyanosed to some degree at least. Because the right ventricular pressure cannot exceed the left, as it does in severe pulmonary stenosis with intact ventricular septum, the degree of right ventricular hypertrophy is less.

The auscultatory signs are a pulmonary ejection murmur and a single second sound.  $P_2$  is inaudible because of the low blood-flow and low pressure in the pulmonary artery.

**Coarctation of the Aorta**

In this condition there is a stricture of the aorta at or near the insertion of the ligamentum arteriosum. In order to maintain a satisfactory circulation to the kidney, the arterial pressure proximal to the coarctation rises considerably and this leads to elongation of the ascending aorta. Prominent arterial pulsation behind the manubrium sterni in a young person strongly suggests this diagnosis, and the carotid and femoral pulses should be felt simultaneously. Normally the timing of the pulse wave is identical. In coarctation the femoral pulse is both delayed and diminished, or it is absent. The arterial pressure is considerably raised; the left ventricle is readily palpable and hypertrophied; there is an ejection click from dilatation of the aorta, and an aortic ejection systolic murmur, often associated with a bicuspid aortic valve. Collateral vessels linking the subclavian arteries which arise above the stricture with intercostal arteries arising below the stricture can usually be felt above the scapulae, and are well seen with the patient bending forwards in a good light (Plate VIII).

**5 Pulmonary Hypertension**

A rise in pulmonary artery pressure to systemic or near-systemic

levels is always associated with narrowing of the pulmonary arterioles either from vasoconstriction or thrombosis and embolism. Pulmonary hypertension may be found in some cases of cor pulmonale, in some cases of septal defect, occasionally with mitral stenosis, or without apparent cause—primary pulmonary hypertension. Whatever the cause, the cardiovascular signs are dominated by the pulmonary hypertension.

There is usually a prominent *a* wave in the neck, the pulse is often very small from the severe obstructive lesion in the pulmonary arterioles, and pulsation over the right ventricle may be palpable. On auscultation there is a loud ejection click, close splitting of the second sound with a very loud and palpable pulmonary valve closure and often a pulmonary diastolic murmur.

## 6 Flow Murmurs

Flow murmurs are due to an abnormally high flow of blood through a normal valve, or to distortion or angulation of the aorta or pulmonary artery beyond the valve.

Ejection systolic murmurs may arise at the aortic or pulmonary valves in cases of anaemia or thyrotoxicosis when the resting cardiac output is considerably raised. In congenital heart disease it will be seen that flow murmurs can arise at the pulmonary, tricuspid or mitral valves, when the blood-flow through these valves is considerably raised in septal defect.

In infants and young children flow murmurs are very common, and the way in which they arise is not fully understood. They usually disappear as the child grows older.

In the adult most flow murmurs are aortic. They are loudest in expiration, best heard in the mitral or aortic area, soft, and never accompanied by a thrill. The other findings in the cardiovascular system are normal, except where the cardiac output is raised. The distinction between trivial aortic stenosis and an aortic flow murmur is difficult.

## 7 Exocardial Sounds and Murmurs

### 1 Venous Hum

Sometimes in children a continuous murmur can be heard in the neck and upper chest which is due to kinking and partial obstruction of one of the larger veins in the neck, and interference with continuous

flow of blood through the vein. The origin of the murmur should be suspected because of the youth of the patient, and the loudness of the murmur in the neck. The hum can be obliterated by pressure on the neck, which produces complete obstruction of the vein, or by altering the position of the neck so as to relieve the venous obstruction. It is particularly important to exclude a venous hum if a diagnosis of patent ductus is being considered.

## 2 Cardio-respiratory Murmurs

These murmurs are systolic and due to the rhythmic compression of a lobule of lung by the beating heart. Characteristically these murmurs are loudest at a particular point in the respiratory cycle, disappearing as the patient breathes in a little more or out a little more.

## 3 Pericardial Friction Rubs

These have a superficial 'leathery' quality and have a to-and-fro character, being present both in systole and diastole. This may be sharply localized and vary in position from day to day. Effusion into the pericardium, by separating the pericardial surfaces, may eliminate the rub. Pleural rubs from pleurisy of a portion of lung near the heart may have a similar quality but are much reduced in intensity by having the patient hold his breath.

# VI ELECTROCARDIOGRAPHY

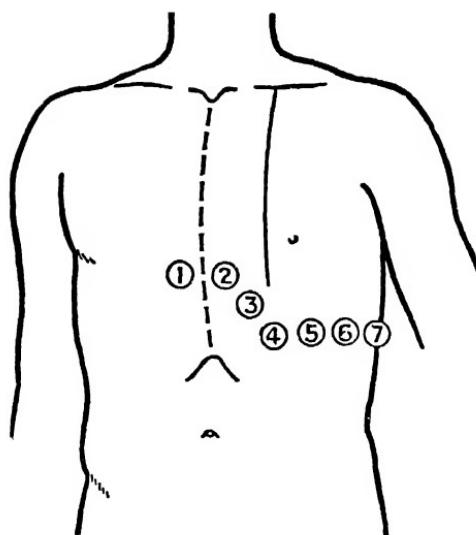
The action of the excitable tissues of the body is associated with electrical activity. Changes in electrical potential associated with the contraction of the heart can be recorded from the body surface.

There must be two points of contact with the body to lead the electrical activity of the heart to the galvanometer. These connections are termed electrocardiographic leads. The leads in common use are:

### **The standard limb-leads (bipolar limb-leads):**

- Lead I, right arm—left arm.
- Lead II, right arm—left leg.
- Lead III, left arm—left leg.

**The unipolar or V leads.** The two connections in these leads are (i) an exploring electrode and (ii) an indifferent electrode which is produced by joining the limb-leads together and thence through a resistance—for all practical purposes it is neutral.



**Fig. 14.** The position of the exploring electrode for chest leads.

**The unipolar limb-leads.** These are termed:

- a VR—the exploring electrode placed on the right arm.
- a VL—the exploring electrode placed on the left arm.
- a VF—the exploring electrode placed on the left leg.

**The unipolar chest-leads.** The position of the exploring electrode is as follows:

- V<sub>1</sub>—4th intercostal space just to the right of the sternum
- V<sub>2</sub>—4th intercostal space just to the left of the sternum
- V<sub>3</sub>—lies midway between V<sub>2</sub> and V<sub>4</sub>
- V<sub>4</sub>—5th left intercostal space in the mid-clavicular line
- V<sub>5</sub>, V<sub>6</sub> and V<sub>7</sub>, lie on the same horizontal line as V<sub>4</sub> in the anterior, mid, and posterior axillary lines respectively.

The deflections or waves of the electrocardiogram are designated by the letters PQRST as shown (Fig. 15A). The P wave is associated with atrial excitation, the QRS with ventricular excitation, and the T wave with ventricular recovery. The Q wave is an initial downward deflection in the QRS complex (Fig. 15B).

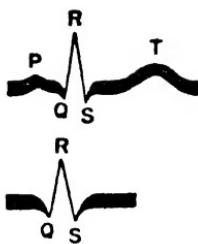


Fig. 15. Terminology of the electrocardiogram.

The PR interval (measured from the beginning of the P wave to the beginning of QRS complex) is normally less than 0·2 secs. The duration of the QRS complex is normally less than 0·1 secs.

A

B

**Axis deviation.** This is a term used to describe the direction of the main QRS deflection in the frontal plane. This depends on the anatomical position of the heart; if the apex is tilted to the left and upwards, *left axis-deviation* is present; if the apex is rotated downwards and towards the mid-line, *right axis-deviation* is present.

The axis-deviation is ascertained from the limb leads.

In *left axis-deviation* there is a taller R than S in lead I and a deeper S than R in III.

In *right axis-deviation* the S is deeper than the R in I and the R taller than the S in III.

It must be appreciated that axis-deviation is of no great diagnostic value. Usually (but by no means always) left axis-deviation accompanies left ventricular hypertrophy and left bundle-branch block, and right axis-deviation accompanies right ventricular hypertrophy and right bundle-branch block.

### The Electrocardiogram in Disorders of Cardiac Rhythm

By providing together a record of atrial excitation (P waves) and ventricular excitation (QRS complexes) the electrocardiogram has advanced our understanding of the cardiac arrhythmias. P waves are usually best seen in Lead II or in the right-sided chest-leads ( $V_1$ ) and these leads are therefore most valuable in the disorders of cardiac rhythm.

In health the heart beat is initiated in the sino-atrial node (pacemaker) which lies near the entry of the superior vena cava into the right atrium. The impulse spreads through both atria and thence to the atrio-ventricular node lying in the inter-atrial septum. The AV (atrio-ventricular) node is continuous with the bundle of His and its branches. The commonest disorders are:

#### 1 Sinus Tachycardia (Fig. 16)

The cardiac impulse arises normally, and the electrocardiogram is

normal in form. The pulse rate is increased above 90 or 100 (adults).



**Fig. 16. Sinus tachycardia.**

Pulse rate 120. Timing marks on this and subsequent electrocardiograms are at 0.2-sec intervals.

Sinus tachycardia may result from emotion, exercise, fever, hyperthyroidism, and anaemia.

## 2 Sinus Bradycardia (Fig. 17)

Again the electrocardiogram is normal in form, but the heart rate is less than 60 per minute. Sinus bradycardia occurs in athletes, and



**Fig. 17. Sinus bradycardia.**

Pulse rate 55.

in patients with increased intracranial pressure, myxoedema and jaundice.

## 3 Sinus Arrhythmia (Fig. 18)

The cardiac impulse arises normally in the sino-atrial node, the rhythmicity of which varies: the heart rate increases with inspiration

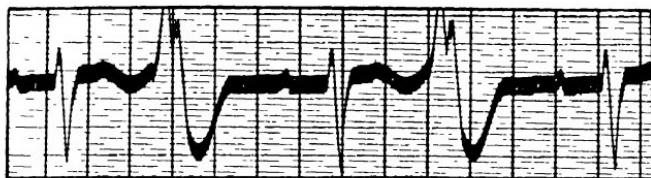


**Fig. 18. Sinus arrhythmia.**

and diminishes with expiration. The electrocardiogram is normal apart from variation in the R-R intervals. This arrhythmia is a normal finding in young people; it is increased by deep breathing and abolished by exercise.

#### 4 Extrasystoles or Ectopic beats (Figs. 19, 20)

These beats arise from foci in the atria or ventricles which stimulate the heart before the next sinus beat is due. In ventricular extrasystoles P waves are absent and the QRS complexes are like those of bundle-branch block, the T wave pointing in the opposite



**Fig. 19. Ventricular extrasystoles.**

Note that there is no P wave before the second extrasystole, and there is an abortive P wave just before the first extrasystole.

direction to the major deflection of the QRS. The rhythm of the sino-atrial node is not disturbed. The extra-systole comes prematurely and is followed by a pause (the compensatory pause).

The electrocardiogram of an atrial extrasystole shows the P wave to be abnormal in form, but the QRS which follows it is normal. The pause which follows the extrasystole is longer than normal.



**Fig. 20. Atrial extrasystoles.**

Note the abnormal (inverted) P wave. The R-R interval is longer following the extra systole than in the next normal cycle.

Extrasystoles are thus premature beats followed by an abnormally long pause and can be recognized by auscultation or from palpation. Extrasystoles occur both in health and in patients with heart disease. If an extrasystole follows after each normal beat as in Fig. 19 the pulse is said to be coupled (*pulsus bigeminus*). Digitalis often causes coupling.

#### 5 Atrial Tachycardia and Atrial Flutter (Fig. 21, 22)

These are due to the presence of an ectopic focus in the atrium which beats regularly at a rapid rate. The P waves are abnormal in shape, but the QRS complexes are normal. As a rule not all atrial impulses



**Fig. 21. Atrial tachycardia.**

During attack—pulse rate 225. After attack—sinus tachycardia—pulse rate 130.

are conducted to the ventricles. Often alternate beats are conducted when 2:1 atrio-ventricular block is said to be present. Occasionally 3:1 or 4:1 block is present and sometimes the block varies.

Atrial flutter and tachycardia may occur in hearts which are other-



**Fig. 22. Atrial flutter, 2 : 1 block.**

Atrial rate about 300. Note two spiked flutter waves to each ventricular complex.

wise normal, in thyrotoxicosis and in rheumatic or ischaemic heart disease.

## 6 Atrial Fibrillation (Fig. 23)

There is no co-ordinated atrial activity (either electrical or mechanical). The electrocardiogram shows *f* (fibrillation) waves representing



**Fig. 23. Atrial fibrillation.**

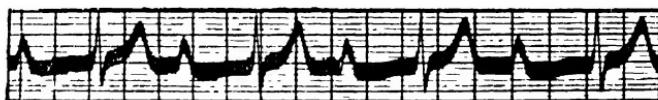
Note '*f*' waves and irregular ventricular rhythm.

the atrial activity instead of P waves, especially in lead V<sub>1</sub>. The QRS complexes are normal, but are irregularly spaced.

Atrial fibrillation is recognized clinically by complete irregularity of the pulse both in rate and volume. Mitral valve disease, ischaemic heart disease, and thyrotoxicosis are the commonest causes of fibrillation.

### 7 Heart Block (Figs. 24, 25)

This may be partial or complete. In the former the P-R interval exceeds 0.22 sec. Sometimes the ventricle responds only to every



**Fig. 24. Partial heart block.**

P-R interval = 0.42 sec.

second, third, or fourth atrial stimulus. In complete heart block the atria and ventricles beat independently, the ventricles having their own pacemaker, which has a slow rate usually 20–40 per min.



**Fig. 25. Complete heart block.**

Auricular rate 55. Ventricular rate 39.

### 8 Ventricular Fibrillation

A similar mechanism operates in the ventricles as in the atria with atrial fibrillation. There are no distinct QRS complexes, but bizarre undulations of the tracing of irregular height and rate.

#### The Electrocardiogram in Other Conditions

The electrocardiogram can give valuable information in conditions other than the arrhythmias. The following account is not intended to

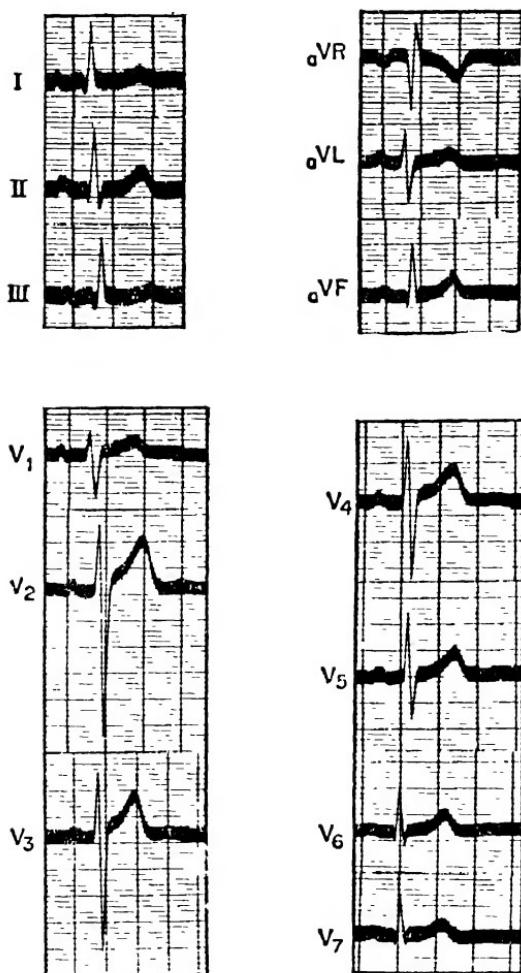


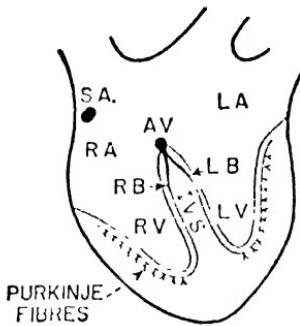
Fig. 26. Normal electrocardiogram.

Standard leads (I II III), V (unipolar) limb leads, V (unipolar) chest leads V<sub>1</sub>-V<sub>7</sub>

be exhaustive, and contains many simplifications. Considerable experience is required even to be familiar with the limits of the normal. It will be noted that many electrocardiographic abnormalities can arise from a variety of causes. The interpretation of an abnormal electrocardiogram depends on the recognition of one of the basic abnormal patterns to be described below and its correlation with the clinical findings.

### 1 Ventricular Hypertrophy

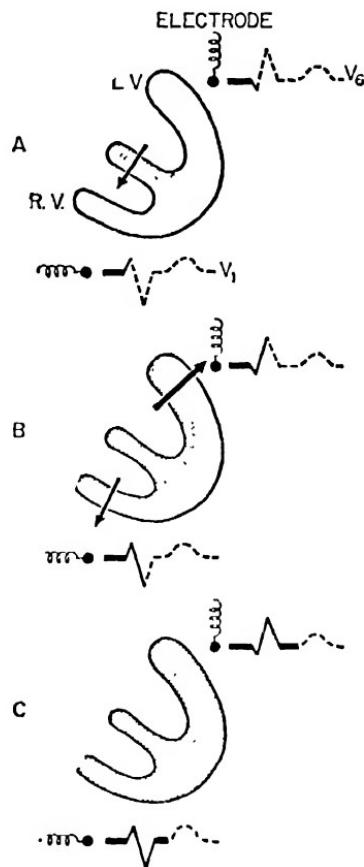
Ventricular hypertrophy is diagnosed principally from the chest leads and in particular  $V_1$  and  $V_6$ . Fig. 29 shows the production of the QRS complexes in  $V_1$  (over the right ventricle) and  $V_6$  (over the left ventricle). It must be appreciated that the QRS complex from any lead represents the algebraic



**Fig. 27. The conduction system of the heart.**

SA = sinus node; AV = atrio-ventricular node; RB = right Bundle of His; LB = left bundle of His.

(After Goldberger.)



**Fig. 28. To show the spread of stimulus through normal ventricular muscle.**

The tracings of a chest lead facing the right ventricle and of a chest lead facing the left ventricle are shown. The shaded areas show stimulated muscle. The arrows show the direction in which the stimulus spreads through the muscle.

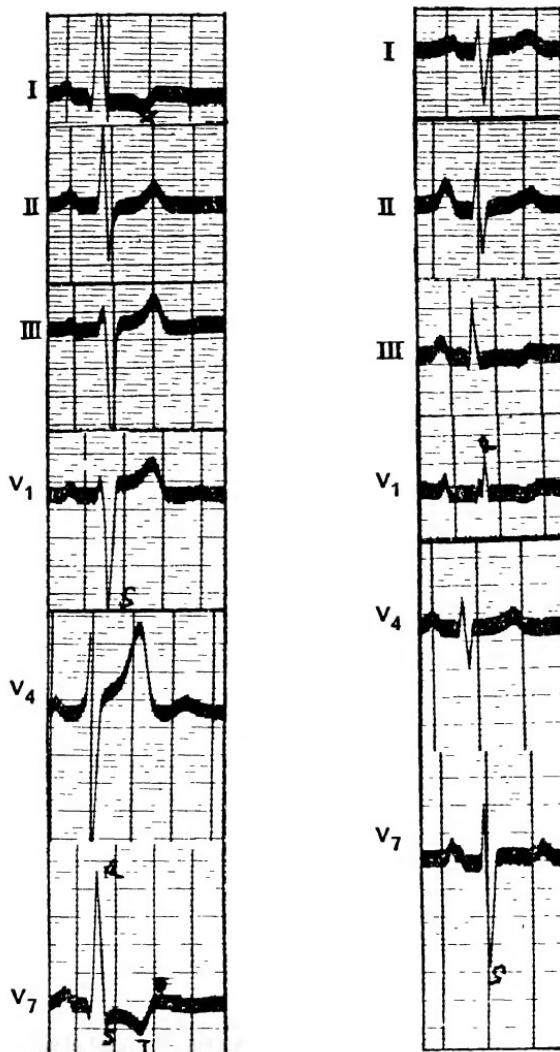


Fig. 29.

**A. Left ventricular hypertrophy.**

Note deep S wave in V<sub>1</sub> and tall R wave in V<sub>7</sub>. Left axis deviation, S-T depression and T wave inversion in lead I and V<sub>7</sub>.

**B. Right ventricular hypertrophy.**

Note tall R wave in V<sub>1</sub> and deep S wave in V<sub>7</sub>. Right axis deviation, T wave inversion in lead III and V<sub>1</sub>. In this tracing the T wave is normal in lead II, though it is usually flat or inverted in right ventricular hypertrophy. The P wave in V<sub>1</sub> is sharp and peaked, suggesting right atrial hypertrophy.

sum of the electrical activity of both ventricles. At any point in time the ECG will show an R wave if the resultant is directed towards the electrode and an S wave if the resultant is going away from the electrode. An additional factor is that the ventricle nearest the electrode will produce a greater deflection than the distant ventricle.

*Left ventricular hypertrophy* (Fig. 29A). As would be expected, the S wave is larger in leads facing the right ventricle and the R wave is larger in leads facing the left ventricle. In addition there is inversion of the T waves and depression of the ST segments in limb lead I (and often II) and in the chest leads facing the left ventricle.

*Right ventricular hypertrophy* (Fig. 29B). The R wave is tall in the leads facing the right ventricle and there is a deeper S than usual in left ventricular leads, i.e. V<sub>5</sub> and V<sub>7</sub>. In addition there is T wave inversion and ST depression in limb leads II and III and in the chest leads facing the right ventricle.

## 2 Bundle Branch Block (Fig. 30)

Owing to an interruption of a branch of the bundle of His conduction to one ventricle is delayed. The QRS duration is greater than 0.1 secs. and the pattern is similar to ventricular hypertrophy. For example, if the left bundle is interrupted, conduction to the left ventricle is delayed and it becomes activated at a time when it is no longer opposed by the right ventricle, and therefore produces larger deflections, as in hypertrophy of the left ventricle.

## 3 Cardiac Infarction

Cardiac infarction alters the electrocardiogram by the production of abnormal Q waves or abnormalities in the S-T segments and T waves, or both. Within a few hours of infarction the S-T segments become elevated (Pardee's sign) (Fig. 31B). In a few days the T waves become inverted, often steeply so (Fig. 31C). The S-T segment gradually returns to the base line, taking several weeks to do so. T wave inversion may eventually return to normal (Fig. 31D), but some inversion usually persists. Abnormal Q waves are usually permanent. The leads showing Q waves or S-T and T wave change are determined by the site of infarct. The electrocardiograms illustrating classical anterior and posterior infarction (Fig. 32A and B) are tracings taken several weeks after the infarction.

In *anterior infarction* the changes are seen in leads I and V<sub>4</sub>.

In *posterior infarction* the changes are seen in leads II and III and aVF.

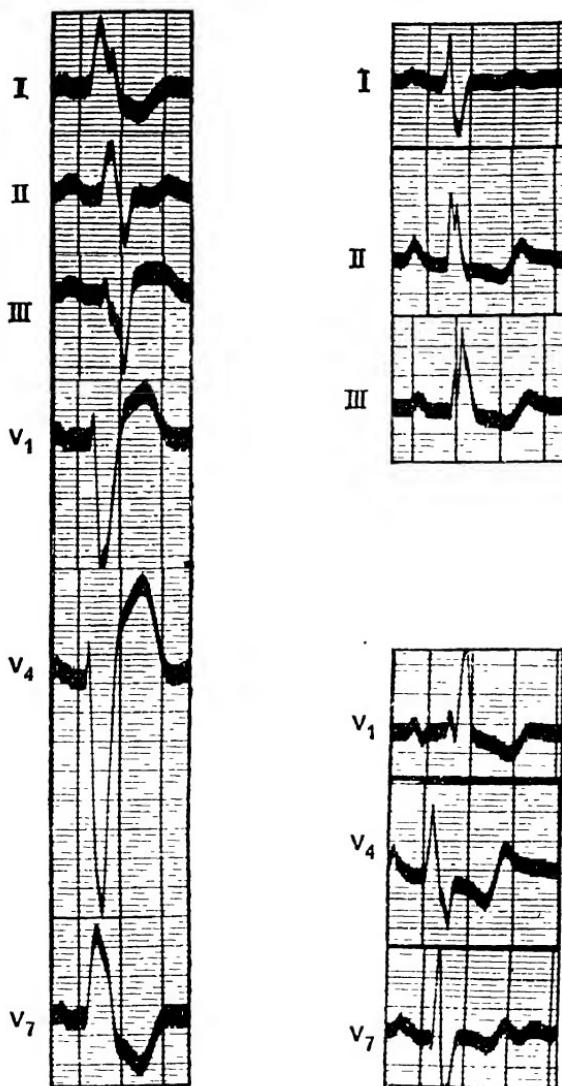


Fig. 30.

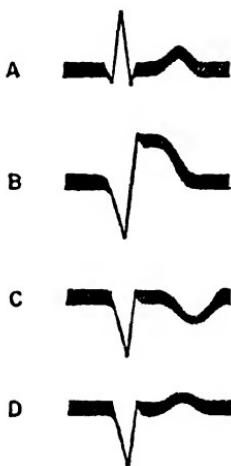
**Left bundle branch block.**

Note wide QRS complexes (0.15 sec.) and deep, wide, slurred R wave in V<sub>1</sub>.

**Right bundle branch block.**

Note wide QRS complexes (0.15 sec.), rsR pattern in V<sub>1</sub> and deep, wide S wave in V<sub>7</sub>.

In *lateral infarction* the changes occur in leads I, AVL and V<sub>5</sub>. In *septal infarction* the changes occur in lead V<sub>1</sub>. An infarct in this area, of course, may involve the conduction tissues, and the ECG pattern may be altered by the appearances of bundle-branch block or complete heart-block.



**Fig. 31. Diagram to show the evolution of changes in the QRS complex, S-T segment and T wave after cardiac infarction.**

- A. Normal pattern.
- B. A few hours after infarction. A Q wave is present. The S-T segment is elevated (Pardee's sign).
- C. After a time the S-T segment returns to the base line and the T wave becomes steeply inverted.
- D. After a further period, the T wave becomes less inverted, flat and finally upright. Note that the Q wave persists.

#### 4 P Wave Changes

In right atrial hypertrophy, due for example to chronic cor pulmonale or pulmonary stenosis, the P wave is tall and sharp. In left atrial hypertrophy, especially in mitral stenosis, the P wave is broad and bifid.

#### 5 S-T segment and T Wave Changes

This occurs in many conditions:

1. Digitalis depresses the S-T segment in all leads; especially in

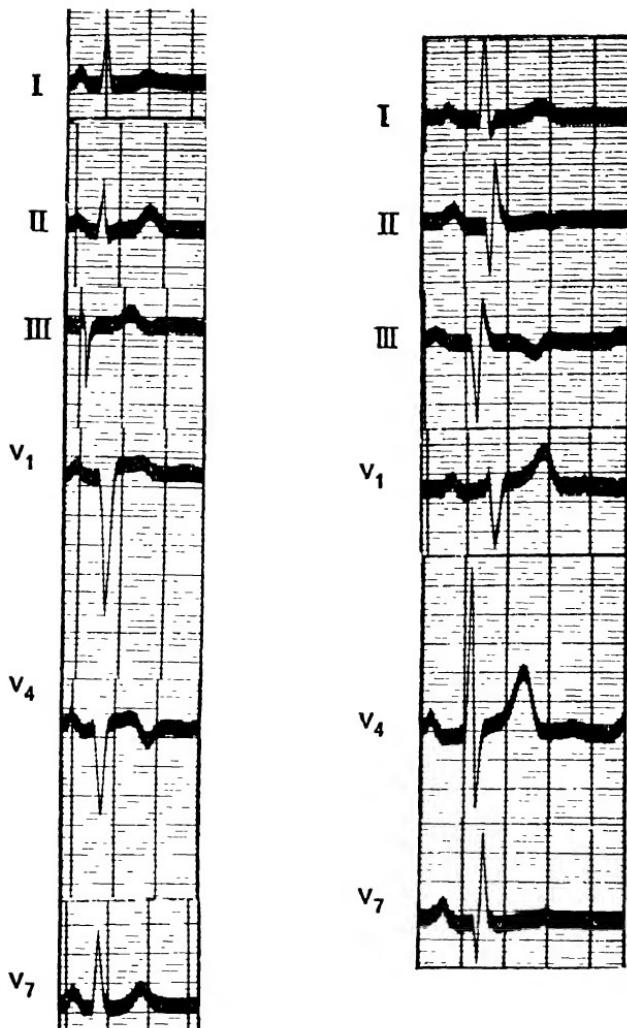


Fig. 32.

**A. Anterior cardiac infarction.**

Note Q wave in  $V_1$  and  $V_4$ . T wave is low in lead I and diphasic in  $V_4$ .

**B. Posterior cardiac infarction.**

Note Q waves in leads II and III. The T wave is slightly inverted in lead II and inverted in lead III.  $V_1$  also shows a Q wave and a low T wave so that this is really postero-lateral infarction.

- limb leads I and II and in the chest leads facing the left ventricle.
2. Flat or inverted T waves occur in myxoedema and are associated with low voltage QRS complexes and bradycardia.
  3. Hypokalaemia causes depression of the S-T segments and inversion of the T waves, while hyperkalaemia causes tall sharp T waves.
  4. Pericarditis causes the S-T segment to be elevated in some leads, especially in lead II. The segment retains its normal concavity. This change is due to epicardial damage and is strongly suggestive of acute pericarditis. In chronic pericarditis there is T wave inversion.

## 6 Ischaemic Heart Disease

In *ischaemic heart disease* (but without a frank cardiac infarction) there may be ST segment depression with low or inverted T waves.

## 7 Prolongation of the Q-T Interval

This occurs in most cases of active rheumatic fever. Often the P-R interval is also prolonged. Digitalis shortens the Q-T interval.

# VII X-RAY EXAMINATION OF THE HEART

In addition to the standard postero-anterior X-ray of the chest much information is gained by taking more penetrated films in the postero-anterior and right lateral views.

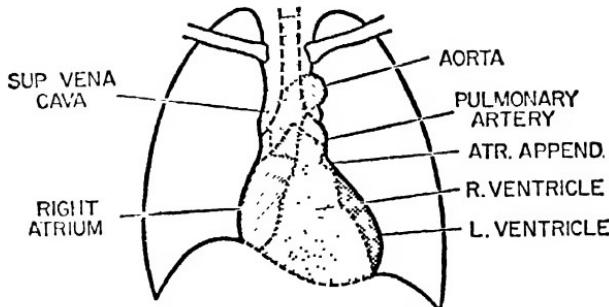
Screening of the heart is occasionally of value in visualizing calcification in valves, the degree of pulsation in the pulmonary arteries or aorta, and the differentiation of pulsatile from non-pulsatile shadows.

### The Normal Cardiac Outline

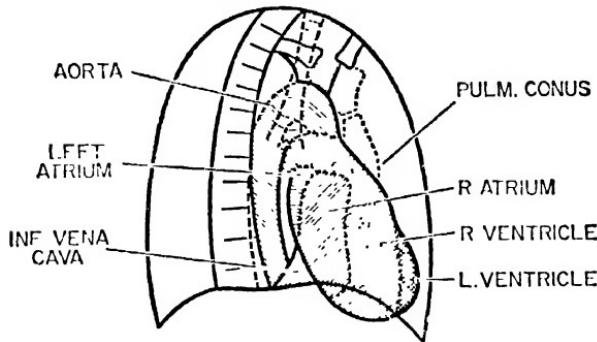
The heart is seen as a flask-shaped shadow, lying between the translucent lungs, about one third of its area to the right of the mid-line and two thirds to the left. The apex of the heart is internal to the midclavicular line.

*The right border* of the cardiac shadow is formed, from above downwards, by two curves:

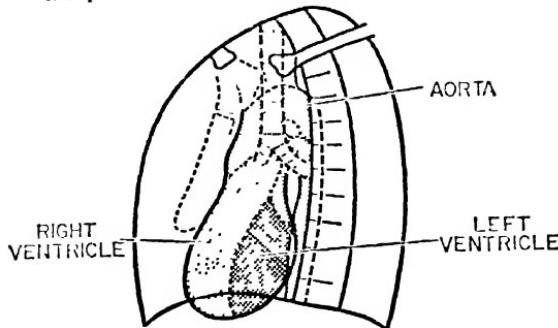
- (i) A slightly curved portion, the outer edge of the superior vena cava with the ascending arch of the aorta.
- (ii) A more convex portion, the outer border of the right atrium, which ends at the diaphragm.



**Fig. 33. The cardiac silhouette in the antero-posterior position.**  
Particularly useful in studying the outflow from left ventricle, the pulmonary artery and the aortic knuckle.



**Fig. 34. The cardiac silhouette in the right oblique position.**  
This position is particularly useful in studying the left atrium, pulmonary conus; first part of aorta and a barium-filled oesophagus.



**Fig. 35. The cardiac silhouette in the left oblique position.**  
Particularly useful for studying the left ventricle, the aortic arch and the descending aorta.

The *left border* is made up by:

- (i) Above, the prominent knuckle produced by the arch of the aorta as it passes backwards, slightly to the left, then downwards.
- (ii) The straighter line of the pulmonary artery.
- (iii) Below, the wide sweep of the left ventricle, ending at the apex, where it rests on the diaphragm.

In the over-penetrated antero-posterior film the left atrium can be seen, especially if enlarged, and the aorta is particularly well shown. Calcification in the pericardium or valves is usually apparent. The right lateral film is also of value in showing calcification and in addition is helpful in right ventricular hypertrophy when the cardiac shadow comes closer to the sternum and higher up the sternum than usual.

### Common Alterations in Disease

#### 1 Position of the Heart in the Chest

Displacement of the heart as a whole is seen in pleural effusion, pneumothorax, and fibrosis of the lung. In distension of the stomach and obesity, the heart is raised with the diaphragm and the apex tilted upwards. The common type of scoliosis (convexity of the curve to the right) is a frequent cause of displacement of the heart to the left. In narrow chests the heart often lies centrally and seems small and slender.

#### 2 Shape and Size of Heart

(a) Prominence and undue convexity of the left ventricle is seen in aortic incompetence, in aortic stenosis and in hypertension. In its typical form the outline may be described as boot-shaped. An enlarged left ventricle is well seen (on screening) in the left oblique view when it extends backwards to cover partly the shadow of the spine.

(b) The left atrium is enlarged in mitral stenosis and mitral incompetence. The left atrial appendix shows in chest X-rays as a straightening or convexity of the normally concave left border of the heart below the pulmonary artery.

The over-penetrated grid film is particularly useful in detecting left atrial enlargement. The outline of the enlarged left atrium can usually be seen through the right atrial shadow on the right border. On the left, the dark shadow of the left main bronchus is displaced upwards by the enlarged left atrium and lies more horizontally.

Left atrial enlargement can also be assessed by giving the patient barium paste to swallow and screening him in the right oblique position. The oesophagus is displaced backwards and to the right by the enlarged left atrium.

### 3 Shape and Size of the Aorta and Superior Vena Cava

Aortic enlargement occurs in syphilitic aortitis with aneurysm. Unfolding of the aorta is seen in atheroma, especially when hypertension is associated. The shadow of the superior vena cava is widened in congestive heart failure.

### 4 Pulsation of the Heart and Aorta

An abnormal degree of aortic pulsation is a feature in the cardio-scopic examination of cases of aortic incompetence. In mitral incompetence, the shadow of an enlarged left atrium is seen in the antero-posterior view to expand markedly during ventricular systole. A saccular aneurysm of the aorta appears as a rounded outgrowth from some part of the aorta, which itself will often be dilated. Its relation to the course of the aorta and its pulsation help to distinguish it from an intra-thoracic tumour.

### 5 The Hilar Shadows and Lung Fields

In mitral stenosis, where the left atrial pressure is significantly raised, horizontal lines (Kerley B lines) due to engorged sub-pleural lymphatics are seen at the lung bases. The hilar shadows are heavy and rather ill-defined. Similar changes are found in left ventricular failure.

When the pulmonary blood-flow is greatly increased, as in atrial septal defect, the main branches of the pulmonary artery are increased in size—‘pulmonary plethora’. This is especially obvious when some of the branches are seen end-on near the hilum. Conversely, in Fallot’s tetralogy the vascular markings in the lung fields are inconspicuous—‘pulmonary oligaemia’.

### Cardiac Catheterization

A flexible radio-opaque catheter is introduced into a vein, and manipulated under fluoroscopic control into right atrium, right ventricle, and pulmonary artery. The technique can be used in several ways:

1. Pressures within the cardiac chambers and vessels can be

measured. For instance, the systolic pressure-gradient across a stenotic pulmonary valve can be measured directly. It is also possible to measure left atrial pressure indirectly, and hence to assess mitral valve disease.

2. Blood samples can be taken from various sites and analysed for oxygen content. The cardiac output can be measured, using the Fick principle—the mixed venous sample being obtained from the pulmonary artery through the catheter, the arterial sample by means of an arterial needle, and the oxygen uptake by analysis of the expired air. Septal defects can be demonstrated by blood-sampling. Let us suppose that samples from the pulmonary artery and right ventricle are 80 per cent oxygenated, while samples from the right atrium and superior vena cava are 65 per cent oxygenated. Oxygenated blood is obviously entering the right ventricle through a ventricular septal defect and the amount of blood flowing through the defect can be calculated.

3. The catheter may pass into abnormal vessels or into the left heart through a septal defect or patent foramen ovale.

4. The left-sided heart chambers may be entered either from the right atrium, by puncturing the atrial septum with a fine needle passed through the catheter, or by inserting a catheter into an artery and passing it backwards in the aorta and left ventricle.

### Angiocardiography

The information obtained from cardiac catheterization is predominantly physiological, e.g. the flow of blood across a ventricular septal defect, or the gradient across a pulmonary stenosis. What the surgeon requires is precise anatomical information, and angiography can provide this. The present trend is for the physiological facts to be deduced by clinical examination, and precise anatomical information to be obtained by angiography.

Angiography consists of the taking of X-ray films while a contrast medium is injected into the heart. To obtain good X-ray pictures, the contrast medium must be highly concentrated in the critical area. It is therefore injected through a catheter, which is placed in the appropriate heart chamber, e.g. in the right ventricle just below a stenotic pulmonary valve, and this is called selective angiography. A rapid injection is obtained by a mechanical pump and an elaborate mechanical film changer takes twelve or more films a second. Cineangiography is an elaboration of this technique.

## **5 CLINICAL EXAMINATION OF THE BLOOD**

### **I EXAMINATION OF STAINED FILMS**

The first step in the examination of the blood is the preparation of stained films. The examination of a properly made blood film by a competent observer, together with a determination of the level of haemoglobin (or of the volume of packed cells), is enough in the majority of cases either to exclude disease of the blood or to suggest a diagnosis. Red-cell counts are tedious and inaccurate, unless electronic methods of counting are available, and such counts are seldom necessary.

Films may be made from anticoagulated venous blood (p. 350). In this case they should be made as soon as possible and not from blood which has been allowed to stand. For most purposes they are best made from a drop of peripheral blood obtained by pricking the lobe of the ear or the finger (or the heel in the case of infants) with a sharp sterile needle. Blood for haemoglobin level and other purposes can be obtained at the same time.

The ear or finger should be pink and warm. The puncture should be made vertically to a depth of 2 to 3 mm. so that a free flow of blood is obtained. Wipe away the first few drops and then take samples when the blood is flowing freely. Squeezing should be avoided. The needle used must be flamed between each patient.

The slides used for making films should be entirely free from grease. To ensure this they should be dropped one by one into an enamelled iron dish containing 10 per cent chromic acid and boiled for 20 minutes. They should then be tipped into a shallow basin, and water allowed to run on them till the washings are colourless. After this they are covered with spirit, and finally transferred with forceps to a wide-necked, stoppered bottle containing absolute alcohol. When required, they should be picked out with forceps, allowed to drain and then dried off with a clean cloth. They should finally be rubbed with a clean handkerchief. Slides may also be satisfactorily cleaned by polishing them with the finest emery paper and then leaving in absolute alcohol until required.

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If slides have to be cleaned in a hurry, glacial acetic acid followed by water and alcohol gives good results.

**How to make films.** Apply one end of a slide to a drop of blood and place the slide on a level surface, holding it with the thumb and index finger of the left hand. The narrow edge of a second slide is placed in the drop and held there till the blood has spread across it; it is then drawn slowly over the whole length of the first slide. The inclination of the second slide to the first should be 45°, and there should be no pressure whatever between the two surfaces. The more slowly one slide is drawn over the other, the thinner is the resulting film. Smooth spreading of the film is aided by warming the first slide in the flame of a spirit-lamp before applying it to the drop of blood. After the blood is spread it should be dried by being waved rapidly in the air to prevent undue shrinkage of the cells.

**How to stain the film.** Either of the two following methods gives excellent results:

1. *Jenner's stain.* (The stain consists of a 0·5 per cent solution of a specially prepared crystalline compound of methylenc blue and eosin in pure methyl alcohol.) Films are made in the usual way. As soon as they are dry, a few drops of the solution are poured on, and they are covered with watch-glasses to prevent evaporation and precipitation of the stain. Pour off in one to four minutes. Rinse in *distilled* water till pink (5 to 10 seconds). Dry rapidly high over a flame or by waving in the air. Mount in xylol balsam. In a successful film the red corpuscles are brownish-red; nuclei are blue, platelets mauve, the granules of polynuclear cells and myelocytes red, basophils dark violet, bacteria, filarial and malarial parasites blue.

2. *Leishman's stain.* This is a simplification of the method of staining first introduced by Romanowsky. The dry film is well covered with the stain, which should be evenly distributed over the entire slide or cover-glass. At the end of one minute, double the quantity of distilled water is carefully added and mixed with the stain. At the end of seven minutes, the mixture is poured off, and the film covered with distilled water for two minutes. The water is then washed off with fresh distilled water, and the film gently blotted dry with clean blotting-paper. When dry it can be mounted in xylol balsam, or examined directly under the oil-immersion lens.

**Examination of the film.** First make a general examination of the

film. This is best done by placing a drop of mountant on the film, covering it with a cover-glass and using the low power (16-mm. objective) followed by the high power (4-mm. objective). The oil immersion objective should be reserved for the examination of abnormal cells and fine points of morphology. Observe whether the film is properly spread. Then observe whether red cells, white cells, and platelets are present in about normal proportions, or whether there is a gross excess of white cells, suggesting leukaemia (Plate XVII), or a severe deficiency of platelets, suggesting thrombocytopenia. For parasites in the blood, see plate XIV.

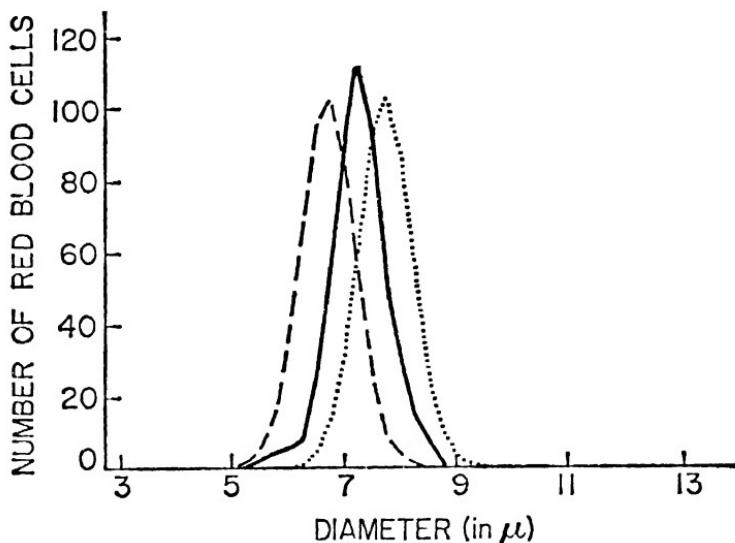


Fig. 36. Red-cell-diameter distribution-curve in a normal woman.

Ideal curve for the smallest ( $-3\sigma$ ) mean diameter within normal limits — — —  
 Ideal curve for the largest ( $+3\sigma$ ) mean diameter within normal limits .....  
 Curve in a case of a normal woman — — —

The observer should then turn his attention to the morphology of the red cells. The points to be observed are:

1. *The size of the red cells.* Normal red cells form a homogeneous population with a fairly narrow variation around a mean diameter. For special purposes red-cell-diameter distribution curves can be constructed. Normal curves are shown in Fig. 36. The mean diameter in health varies from 6.7 to 7.7  $\mu$  with an average of 7.2  $\mu$ . The width of the base of the curve (or more accurately the coefficient of

variability) gives an indication of the degree of inequality in the size of the red cells.

In states of iron-deficiency (Fig. 38) the mean cell-diameter is reduced (*microcytosis*) and the width of the base of the curve is somewhat increased (*anisocytosis*).

In anaemia due to vitamin B<sub>12</sub> deficiency (*pernicious anaemia*), the mean cell-diameter is increased (*macrocytosis*) and the width of the

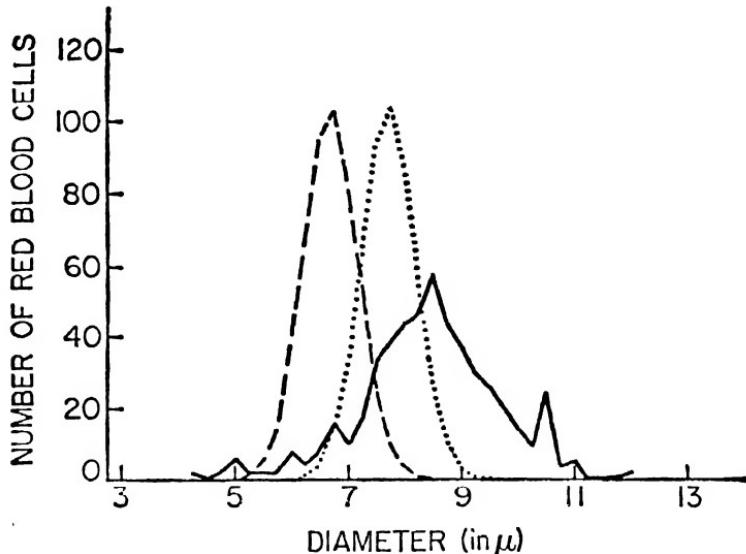


Fig. 37. Red-cell-diameter distribution-curve in a case of pernicious anaemia.

Ideal curve for the smallest ( $-3 \sigma$ ) mean diameter within normal limits — — —

Ideal curve for the largest ( $+3 \sigma$ ) mean diameter within normal limits ..... ·····

Curve in a case of pernicious anaemia — — —

base of the curve may be greatly increased (*great anisocytosis*) (Fig. 37).

Variation in the shape of the red cells, usually the presence of small misshapen cells, is referred to as *poikilocytosis*. It is seen in any severe anaemia, but particularly in pernicious anaemia and haemolytic anaemias.

2. *The staining of the red cells.* Red cells *in vivo* are biconcave discs, and even when fixed and stained they are usually paler at the centre than at the periphery. Poor staining of the red cells (*hypochromia*), along with microcytosis, is characteristic of states of iron deficiency (Plate XVI). In longstanding iron-deficiency virtually all the cells are

hypochromic, whereas with the recent development of iron-deficiency, as from recent haemorrhage, a mixed population of normal cells and hypochromic microcytes is found. The presence of unduly large well-stained red cells, without a pale centre, along with considerable anisocytosis and poikilocytosis is characteristic of anaemia due to vitamin  $B_{12}$  or folate deficiency. Increased segmentation of the nuclei of neutrophils is also present in such circumstances.

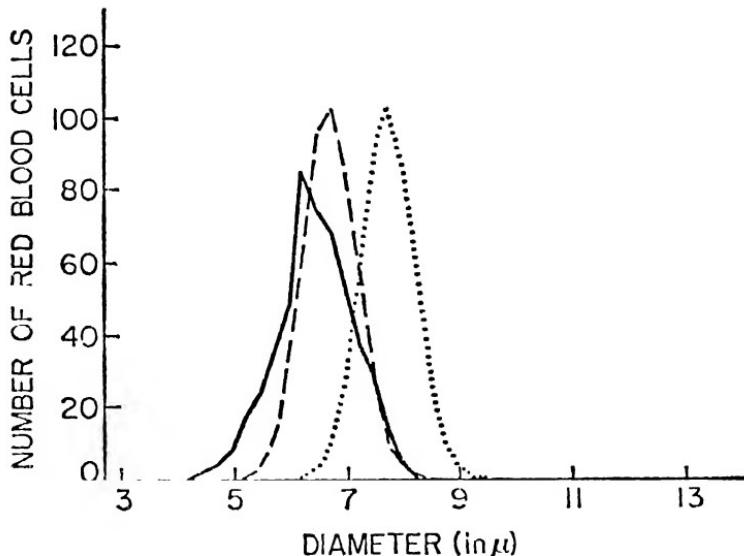


Fig. 38. Red-cell-diameter distribution-curve in a case of iron-deficiency anaemia.

Ideal curve for the smallest ( $-3\sigma$ ) mean diameter within normal limits ————  
 Ideal curve for the largest ( $+3\sigma$ ) mean diameter within normal limits .....  
 Curve in a case of idiopathic hypochromic anaemia —————

(Plate XVI). Mixed pictures of macrocytosis and microcytosis with hypochromia are not uncommon in anaemia caused by small-intestinal disease.

The presence of large numbers of cells showing diffuse bluish coloration (*polychromatophilia*) and speckled blue discoloration (*punctate basophilia*) suggests either intense regeneration (i.e. after haemorrhage or after the administration of a needed specific haemopoietic agent) or some type of haemolytic anaemia. Marked punctate basophilia is characteristic of lead poisoning.

The presence of a number of unusually deeply-stained round cells

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of about normal size (*spherocytes*) together with polychromatophilia and punctate basophilia is characteristic of hereditary spherocytosis (*familial acholuric jaundice*) (see Plate XVI).

Unduly flat cells, which are the opposite of spherocytes, show colour at the periphery, a pale ring and colour again at the centre. They are described as *target cells*, and are found particularly in familial anaemias, associated with the presence of abnormal haemoglobins (*thalassaemia and sickle-cell anaemia*). *Sickle cells* themselves may be seen in the blood film together with target cells in sickle-cell anaemia, owing to the presence of the abnormal haemoglobin S (see Plate XVI).

3. *The presence of nucleated red cells.* Normoblasts can be distinguished from lymphocytes (for which they are apt to be mistaken at first glance) by (i) their more homogeneous and dense dark staining of the nucleus, and (ii) their red cytoplasm.

Megaloblasts are larger cells with a characteristically stippled nucleus and polychromatophilic cytoplasm.

Nucleated red cells (usually normoblasts) may be found in any severe anaemia and in states of intense blood regeneration. They are also found in leukaemia. Megaloblasts are occasionally seen in the peripheral blood in conditions in which they are abundant in the bone marrow (pernicious and other megaloblastic anaemias), but this is an inconstant finding. The presence of a number of primitive red and primitive white cells in the peripheral blood is described as *leuco-erythroblastic anaemia*, and is due in most instances to infiltration of bone-marrow by leukaemia, carcinoma, myeloma cells, or by fibrous tissue (as in myelofibrosis).

## II ESTIMATION OF HAEMOGLOBIN

Accurate haemoglobinometry is now carried out by photo-electric methods, which eliminate errors due to matching colours with the eye. Their accuracy is of the order  $\pm$  2 per cent. Where these are not available, however, Haldane's and Sahli's modifications of the original Gower's dilution and colour-matching method provide the most convenient alternative.

1 **Haldane's haemoglobinometer.** This method uses as a standard of comparison a glass tube containing carboxy-haemoglobin: 100 per cent on the scale corresponds to 14.8 g. of haemoglobin per 100 ml. The colour of the standard is fairly permanent if it is kept in the dark.

The method is as follows:

0·4 per cent ammonia solution (4 ml. liq. ammon. sp. gr. 0·880 in a litre of distilled water) is placed in the diluting tube up to the 20 mark. Water may be used if no ammonia is available, and if the patient is very anaemic the tube should be filled only to the 5 or 10 mark. An adequate puncture is then made in the finger or the lobe of an ear, and the capillary pipette provided filled to the 20 c. mm. mark without delay. The skin and the pipette must be perfectly clean and dry. The pipette should be held horizontally and the point dabbed on the observer's finger till the column of blood is exactly at the 20 mark, and then wiped clean of any surplus blood. The blood is then gently blown into the diluting tube, where it should sink without mixing appreciably with the fluid in the tube; the pipette is washed out several times with this fluid and withdrawn. The solution is then saturated with carbon monoxide with a capillary pipette or lumbar puncture needle, connected by rubber tubing to the gas mains. The gas may be bubbled through the solution or passed over it, while the tube is slanted to expose the greatest fluid surface to the gas. Distilled water is now added drop by drop from a drop-bottle or pipette. After each addition, the diluting tube is held between the thumb and forefinger and inverted to ensure thorough mixing. Any fluid left on the thumb must be wiped back into the tube. Water is added in this manner until the tint in the diluting and standard tubes appears identical. The tubes should be matched in daylight (but not direct sunlight) against a northern sky or against white paper at an angle of 45° or against an electric bulb viewed through an opal-glass screen. It is absolutely necessary to transfer the two tubes repeatedly, while they are being matched, or avoidable error will be made.

The level on the graduated tube is read one minute after the last drop of distilled water has been added. The observation is repeated after the addition of another drop, and if necessary another, until the point is reached when the tints are again just unequal and the average of the two readings is accepted as the final result.

Even among skilled observers, the error of this method may be considerable. Given careful attention to the details of technique and a good eye for matching tints, the error should not exceed 5 per cent. In practice a difference of 5 per cent between two observations by the same observer will usually indicate a real change in the patient rather than an error in the method, and a difference of 10 per cent will nearly always do so. Differences of less than 5 per cent between two observations by the same observer and of less than 10 per cent between

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two observations by different observers should be disregarded.

**2 Sahli's haemoglobinometer.** This method is even less accurate, but has the advantage that, to determine whether a person is significantly anaemic or not, it can be used where no gas is available. It employs as a standard of comparison a glass tube containing acid haematin, the haemoglobin in the diluting tube being converted to the same substance by the addition of hydrochloric acid. In some models a permanent coloured glass standard is employed.

Instruments can be obtained calibrated so that 100 per cent is equivalent to 14 g. haemoglobin per 100 ml. when the reading is made 5 minutes after the addition of the blood to the acid in the diluting tube.

The method is as follows:

The diluting tube is filled to the 10 or 20 mark with decinormal hydrochloric acid. 20 c. mm. of blood are taken up with a pipette as described above and blown gently into the tube, the pipette being rinsed out several times with the acid solution. The tube is allowed to stand for exactly five minutes. Then adding water and matching the tints is carried out as with the Haldane instrument, except that the fluid in the tube is stirred with a small glass rod after each addition of water, instead of the tube being inverted.

The error of this method is greater than that of the Haldane, and the same precautions should be used in interpreting the results recorded. The difference between the Haldane scale (100 per cent - 14.8 g.) and the scale of the Sahli instruments (100 per cent - 14.0 g.) may be neglected. It goes without saying, however, that any report on haemoglobin percentage should state what is meant by 100 per cent in terms of g. per 100 ml.

The normal haemoglobin varies within considerable limits and differs in the two sexes and at different ages. The mean haemoglobin of healthy men may be taken as 15.6 g. per 100 ml., equivalent to 113 per cent in the Haldane scale, and the normal range from about 100 to 120 per cent. The mean figure for healthy women may be taken as 13.7 g. per 100 ml., equivalent to 99 per cent on the Haldane scale, and the normal range from about 90 to 110 per cent. At birth the haemoglobin may be 140 to 150 per cent; it falls during the first few weeks to a level of 70 or 80 per cent, and increases slowly during infancy and childhood.

### III PACKED-CELL VOLUME

This is determined by centrifuging anticoagulated blood in a graduated tube until the corpuscles are packed down to a constant volume. The volume of packed cells is then expressed as a percentage of the original column of blood. Venous blood may be anticoagulated with sequestrene (EDTA) or, if more accurate results are required, an ammonium-potassium oxalate mixture may be used which produces no shrinkage of cells; (5 ml. blood from a vein is placed in a tube containing 4 mg. solid potassium oxalate and 6 mg. solid ammonium oxalate.) With the aid of a capillary pipette, a Wintrobe's haematoctrit tube (which can also be used for determining the sedimentation rate, p. 134), is then filled to the 100 mark with the anticoagulated blood, and centrifuged at 2500 revolutions per minute for 30 minutes. As the original column of blood in the tube is 100 mm. long, the volume of packed cells can be read directly as a percentage. This estimation is simple and accurate, and from this and the haemoglobin the mean corpuscular haemoglobin concentration (MCHC), the most important of the absolute indices, is determined (see p. 126).

### IV RED CELL COUNTS

As already stated, this procedure as performed by the usual methods is tedious, inaccurate and rarely necessary. Electronic methods of counting are becoming available; but if required, an approximate count can be made by visual methods. The instruments necessary are a graduated mixing pipette and a counting-slide. Either venous blood (p. 350) or peripheral blood (p. 111) may be employed. In the former case the bottle or tube must be well shaken before a sample is taken, and in the latter the sample must be taken from a drop of blood which flows freely without squeezing. Slowly suck up blood with the pipette till the mark 0·5 is reached. If one should go on a little beyond the 0·5 mark, the point of the pipette should be dabbed once or twice on the finger, till the blood is back at the 0·5 mark. Remove any surplus blood from the point, taking care that the column within the pipette does not move, and plunge it at once into the diluting fluid, which should be standing ready in a small wide-necked bottle. Suck up the diluting fluid as far as the mark 101. While this is being done, the pipette should be gently rotated to start the mixing. Grip the pipette firmly by its ends between the forefinger and thumb, and shake thoroughly

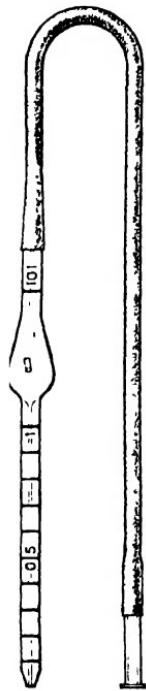


Fig. 39.

**Haemocytometer pipette.**

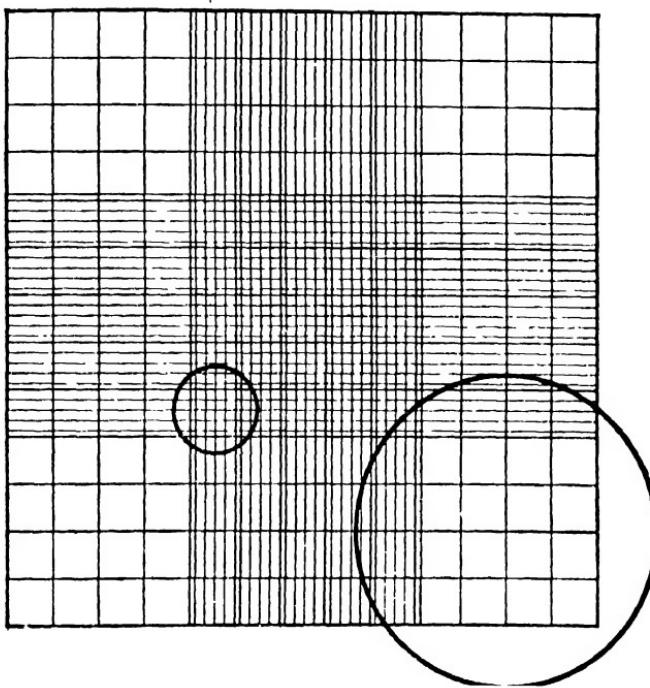
for at least one minute. This ensures a thorough mixing of the blood with the fluid. The column of diluting fluid which occupies the capillary part of the pipette does not enter into the mixture. Hence, if blood is sucked up to 0·5, the solution produced is in the proportion of 1 in 200. The diluting fluid in the capillary tube should now be blown out.

The counting chamber consists of a thick glass slide, with a transverse bar at its centre, the surface of which is sunk  $\frac{1}{10}$  mm. below that of the slide. The bar is separated from the remainder of the slide by two transverse grooves, running parallel to it, one on each side, and is divided at its centre by a further groove, so that two preparations may be set up at the same time. The surface of the bar is ruled with two sets of small squares, each small square having an area of 1/400th sq. mm. A specially ground thick cover-glass (ordinary cover-slips must not be used) is applied to the glass slide over the bar. If the slide and cover-glass are clean and properly applied, concentric colour (Newtonian) rings can be seen, when one is looking almost horizontally along the surface of the cover-glass. The space left between the under surface of the cover-glass and the upper surface of the bar is then exactly  $\frac{1}{10}$  mm. in depth.

When the haemocytometer pipette has been thoroughly shaken, a few drops of the contents of the bulb are blown out and discarded. The pipette is then held at an angle of about 45° to the surface of the counting chamber, and its point applied to the narrow slit between the counting chamber and the cover-slip. The fluid runs under the cover-slip by capillary attraction. This manoeuvre requires some practice. Bubbles must be avoided and the fluid must exactly fill the space between the bar and the cover-slip. If any fluid overflows into the grooves, the counting chamber and cover-slip must be cleaned and the whole operation repeated.

When the counting chamber has been successfully filled, the preparation is set aside for two minutes or so, for the corpuscles to settle. It is then examined with the low power to see whether any air-bubbles or foreign bodies are present, and whether the corpuscles are distributed with fair uniformity throughout the field, after which

the high power (No. 2 eye-piece and  $\frac{1}{2}$ -in. objective) is used for counting. The microscope must be vertical and should be provided with a condenser and a diaphragm. The light should be gradually cut off until the red cells become clearly visible. Under the  $\frac{3}{4}$ -in. objective the little squares will be seen to be marked off into sets of sixteen by double ruling. (Should the lines marking off the squares be only dimly



**Fig. 40. Counting slide, Neubauer ruling.**

The small circle shows the view seen under the 1<sup>st</sup> objective, used in counting red cells. The cells present in 5 sets of 16 small squares (marked off by double ruling) are counted. This number of cells multiplied by 10,000 equal the number of red cells per c. mm. of blood.

The large circle shows the view under the 3<sup>rd</sup> objective used in counting white cells. The cells present in the four large squares (each made up of 16 smaller squares) at the four corners of the ruling are counted. This number of cells multiplied by 50 equals the number of white cells per c. mm. of blood.

seen, it may be necessary to intensify them. This is best done by rubbing the surface of the platform with a little finely powdered graphite - the scrapings from a very soft lead pencil - and then polishing it with soft chamois leather.)

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At least four sets of sixteen squares should be counted under the  $\frac{1}{6}$ -in. objective. The squares in each set should be gone over systematically in horizontal rows of four at a time. Of the corpuscles which lie upon the lines bounding the row, only those on the upper and the left-hand lines should be counted. The number of corpuscles in each of the four sets should be approximately equal.

**Calculation.** Count the corpuscles in each of the four horizontal rows from above downwards. The total is the number of corpuscles in sixteen squares. Count in this way five sets of sixteen, and divide the total by eighty, which gives the average of corpuscles in one square. But the dimensions of this square are

$$\frac{1}{400} \times \frac{1}{10} = \frac{1}{4000} \text{ c. mm.}$$

Therefore, if there are  $x$  corpuscles in this dimension there will be  $4000x$  in 1 c. mm. But the blood was diluted 200 times. Therefore in 1 c. mm. of blood there will be  $4000x \times 200$  corpuscles.

Suppose, for example, that one finds a total of 480 corpuscles in the eighty squares. This gives an average of six corpuscles per square, or  $6 \times 4000$ ; i.e. 24,000 per c. mm. of *diluted* blood, or 4,800,000 per c. mm. of pure blood, if the dilution was 1 in 200. Thus if a counting chamber with the Neubauer ruling (Fig. 40) is used and five sets of sixteen small squares (or eighty small squares) are counted, the addition of four noughts to the figure for the number of red cells in eighty small squares will give the number of red cells per c. mm. of blood.

The normal concentration of red cells varies within considerable limits. At birth it is from 6,000,000 to 7,000,000 per c. mm., and falls during the first week of life to between 4,000,000 and 5,000,000 per c. mm. In adult life it varies in the two sexes. In men the average figure is 5,500,000 per c. mm. The range in health is from about 5,000,000 to 6,500,000 per c. mm. In women the average figure is 4,800,000 per c. mm. and the limit in health about 4,000,000 to 5,500,000 per c. mm. As the error in routine single red counts is up to 500,000, differences of less than this between two counts should be ignored; and preferably all counts should be expressed to the nearest half-million.

## V WHITE CELL COUNTS

A special pipette is supplied for this purpose with the Thoma-

Zeiss instrument. It is used in the same way as the red-corpuscle pipette, but the blood is less dilute. The best diluting fluid is one containing 1 ml. of glacial acetic acid in 100 ml. of water, to which enough of a watery solution of methyl green or gentian violet has been added to give the mixture a decided colour. This mixture dissolves all the red cells while it stains the nuclei of the white.

A large drop of blood should be allowed to exude before the pipette is filled. The blood should be sucked up to the mark 0·5, the end of the pipette wiped, and diluting fluid taken up to the mark 11.

Owing to the relatively large calibre of the pipette, the blood is apt to run out of it, and so the pipette should be kept in a horizontal position as soon as it is filled with blood.

The blood and fluid are mixed as already described. This produces a dilution of 1 in 20. A drop is then placed on the counting-slide with the same precautions as in the case of the red cells.

The leucocytes in the whole of the cross-ruled area of the Thoma counting-chamber, which contains 400 small squares of 1 sq. mm. should be counted. Once one has learned to distinguish leucocytes from other objects, this can be done under the  $\frac{1}{3}$ -in. objective. Several preparations should be made, and the total counts averaged, unless one has a special counting chamber (e.g. Neubauer ruling, Fig. 40) which provides several blocks of 1 sq. mm. The calculation is made as follows. The number of white cells counted in one sq. mm. is that present in  $\frac{1}{10}$  c. mm. of the diluted fluid. This figure multiplied by 200 gives the white cells per c. mm. of blood.

In leukaemia, where a very large excess of leucocytes is present, one can easily count the red and the white cells in the same drop. For this purpose a 3 per cent solution of sodium citrate just coloured with gentian violet is to be preferred for diluting the blood. This stains the nuclei of the whites, and at the same time preserves the reds. The dilution and calculation are the same as for the red cells.

After use, the diluting pipettes should be thoroughly cleaned. They should be washed out (1) with distilled water, (2) with absolute alcohol, and (3) with ether. A stream of air should then be blown through till the glass ball in the chamber moves freely without tending to adhere to the sides. A few drops of antiformin sucked into the pipette will quickly disintegrate any organic matter left behind. Coagulated blood may be removed from the capillary tube with a horse-hair. If the blood adheres firmly to the pipette, it may be removed by repeated rinsing with strong alkali or acid, or if necessary digested away with pepsin.

*The concentration of leucocytes in normal blood is about 7000 per c. mm.*

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in the adult. The number varies within considerable limits even in health, and counts of from 3000 to 12,000 are not necessarily abnormal. In early childhood much higher numbers are reached.

An absolute increase in polymorphonuclear leucocytes is referred to as *leucocytosis* and of lymphocytes as *lymphocytosis*. Apart from leukaemia, which will be mentioned later, important causes of *leucocytosis* are septicaemia and pyaemia, the presence of pus or an abscess anywhere in the body, any infection with pus-forming organisms, such as peritonitis, pneumonia, meningitis, and tonsilitis. Leucocytosis may also be found after acute haemorrhage, in amoebic hepatitis, two or three days after a coronary thrombosis, and occasionally in the presence of malignant neoplasms, particularly of the liver. An absolute *lymphocytosis*, apart from leukaemia, is a much rarer finding. It occurs particularly in glandular fever or infectious mononucleosis, and after whooping-cough.

## VI THE DIFFERENTIAL WHITE CELL COUNT

A well-spread film is essential. It is useless to attempt a differential count if the white cells are congregated in trails and clumps in the tail of the film. Given a reasonably well-spread film, the edges should be avoided and the cells running in strips the whole length of the film should be examined under either the 'high-power' or the oil immersion lens until 200 have been counted.

The following are the varieties of leucocytes found in normal blood, with their relative proportions:

1. *Neutrophil polymorphonuclears*. Cells with multilobed nucleus and fine neutrophil or faintly eosinophil granules: 50–70 per cent.
2. *Eosinophil polymorphonuclears*. Cells with multilobed nucleus and coarse, strongly eosinophil granules: 1–4 per cent.
3. *Basophil polymorphonuclears* (or mast cells). Cells with very pale cytoplasm, a nucleus usually bilobed, and coarse basophil granules 0–1 per cent.
4. *Monocytes*. Cells with a characteristic notched or kidney-shape nucleus and a slightly basophilic, faintly reticular cytoplasm: 2–8 per cent.
5. *Large lymphocytes* with round nucleus and clear basophilic cytoplasm: 5–15 per cent.
6. *Small lymphocytes* with round, deeply staining nucleus which almost fills the cell, leaving a rim of strongly basophilic cytoplasm: 15–40 per cent.

The changes in these cells in *leucocytosis* have been mentioned (p. 124).

In the lymphatic form of leukaemia there is an enormous increase in the number of the lymphocytes (Plate XVII).

In the chronic myeloid form of the disease the neutrophils, eosinophils and basophils are all increased, and in addition bone-marrow cells—myelocytes—appear in the blood. These are often of large size, with a single round nucleus, and contain granules which may be either neutrophilic or eosinophilic in reaction (Plate XVII).

A relative diminution of the leucocytes is spoken of as *leucopenia*. As a rule in leucopenia the diminution affects chiefly the granulocytes, hence the name *granulocytopenia*. There is, of course, a relative lymphocytosis. Leucopenia occurs in:

- (1) Infections such as typhoid fever, undulant fever, and measles.
- (2) Defective bone-marrow function, as in aplastic anaemia or pernicious anaemia.
- (3) Crowding of normal cell production by malignant cells, as in leukaemia or by fibrous tissue, as in myelofibrosis.
- (4) Sensitivity to many drugs, e.g. sulphonamides, thiouracil, amidopyrine, phenylbutazone, chloramphenicol.

It is often necessary also to calculate the *absolute* number of each kind of white cell per c. mm. of blood, as otherwise a relative increase or diminution of one kind may be mistaken for an absolute increase or reduction. Throughout *adult* life the absolute number of polymorphonuclears per c. mm. is 2500–9000, whilst that of the lymphocytes is 1500–4000.

## VII PLATELET COUNTS

The platelets or thrombocytes are elliptical or circular bodies with basophilic cytoplasm and azurophil granules. There is approximately one platelet to every twenty red cells in normal conditions. An accurate platelet count may be performed by using an electronic particle counter, or by indirect phase-contrast microscopy on a Neubauer counting-chamber, using a diluting fluid containing urea, which haemolyses red cells. An approximate platelet count can be performed as follows:

The skin of the ear is cleaned with ether and on the clean surface is deposited a large drop of diluent (3 per cent sodium citrate in normal saline). The skin is then stabbed with a sharp sterile needle so that the

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blood oozes directly into the diluent. This prevents clumping of the platelets. With a platinum loop of 3 mm. diameter some of the diluted blood is transferred to a slide and carefully covered with a cover-slip. The amount taken should be sufficient to spread out evenly between the slide and cover-slip without causing the latter to float. The preparation is ringed with petroleum jelly. Using a microscope fitted with moving stage, squared eyepiece, and  $\frac{1}{10}$ -in. oil immersion objective, the number of platelets and red cells is counted in several fields, thus determining the ratio of platelets to red cells. Knowing the red cell count the actual number of platelets per c. mm. is calculated.

The normal platelet count is from 200,000 to 450,000 per c. mm. However, technical factors such as the development of small clots in the blood-sample may give misleading low counts, and an unexpected low count should always be repeated. Figures below 150,000 certainly constitute *thrombocytopenia*. It may be found in many conditions, including idiopathic thrombocytopenic purpura, aplastic anaemia, leukaemia, pernicious anaemia, and drug reactions. It may be associated with bleeding into the skin and mucous membranes. A real increase in platelets, with counts persistently above 450,000 constitutes *thrombocythaemia*. It may be seen in polycythaemia vera and after splenectomy, and may be accompanied by a tendency to intravascular thrombosis, though an excess of platelets in polycythaemia vera is sometimes associated with a tendency to haemorrhage.

## VIII THE ABSOLUTE VALUES AND THE COLOUR INDEX

**1 Mean Corpuscular Haemoglobin Concentration (MCHC).** This is the most important of the absolute values, as it may be determined with great accuracy and will demonstrate the commonest haematological abnormality—defective haemoglobin production due to iron deficiency.

$$\text{MCHC \%} = \frac{\text{Hb in g. per 100 ml. of blood}}{\text{Volume of packed red cells in c. mm. per 100 c. mm. of blood}} \times 100$$

The normal MCHC is 32–38 per cent. It cannot increase above 38 per cent as the red cell stroma cannot hold a greater than normal concentration of haemoglobin, but reduction below 32 per cent, in the great majority of cases, may be regarded as an indication for iron therapy.

## 2 Mean Corpuscular Volume (MCV)

$$\text{MCV} = \frac{\text{Volume of packed cells in ml. per 1000 ml. of blood}}{\text{Red cells in millions per c. mm.}}$$

This value depends on the red cell count and hence is less accurate than the MCHC. The normal MCV is  $78\text{--}94 \mu^3$ . The value is raised in macrocytic anaemia and reduced in hypochromic microcytic anaemia.

## 3 Mean corpuscular haemoglobin (MCH)

$$\text{MCH in picograms (pg)} = \frac{\text{Hb in g. per 1000 ml. of blood}}{\text{Red cells in millions per c. mm.}}$$

Like the above, this also depends on the red cell count and hence is of limited value. The normal MCH is 27–32 pg. It is usually raised in macrocytic anaemia and reduced in hypochromic anaemia.

**4 The colour index.** The older determination based on haemoglobin and red-cell count is the colour index. It is now largely supplanted by the above. It expresses the mean haemoglobin content of a single cell as compared with that of an arbitrary normal cell. It is obtained by dividing the haemoglobin expressed as a percentage of normal (100 per cent = 14.5 g. of haemoglobin) by the red cell count expressed as a percentage of normal (assumed for convenience to be 5,000,000). Thus if the haemoglobin is 40 per cent and the red cell count is 4,000,000 the colour index is

$$40 \div \left( \frac{4}{5} \times 100 \right) = \frac{40 \times 5}{4 \times 100} = 0.5.$$

More simply it is calculated by doubling the first two figures of the red cell count and dividing the product into the figure for the percentage of haemoglobin. The normal colour index by definition is 1.0 but figures from 0.85 to 1.15 are not necessarily abnormal. The colour index is useful as it divides anaemias approximately into three classes, those with a high colour index, including pernicious and similar anaemias; those with a colour index, about unity, such as anaemia following a recent large haemorrhage, and those with a low colour index such as the iron-deficiency anaemias.

It is subject to the disadvantage, however, that it depends on the accuracy of red-cell counts, which is doubtful, and that it is based on

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arbitrary standards of normal, which take no account of cell size. For this reason the 'absolute' values are preferable.

### IX THE RETICULOCYTE COUNT

The reticulocytes are the youngest cells in the blood. By supravital staining, that is by the application to fresh blood of a special dye before the use of a fixative, they are seen as cells slightly larger in diameter than a mature red cell, containing a delicate basophil cytoplasmic network, which disappears later as the cell matures. The normal reticulocyte count is from 0·5 to 2·0 per cent. The count is increased in states of active regeneration, i.e. after haemorrhage and after a specific haemopoietic remedy has been administered in a deficiency state; and also in haemolytic conditions.

The best dye to use for supravital staining of the blood is brilliant cresyl blue. Saturate 0·85 per cent sodium chloride solution with the dye, filter through a double filter-paper and centrifuge. Pour off the supernatant dye solution and keep it in a stock bottle. For use dilute a small quantity with four volumes of 2 per cent sodium citrate in physiological saline. Puncture the ear and draw a large drop of blood into a Wright's pipette. Follow up the blood by an equal volume of dye. Blow out on to a slide, mix thoroughly, take up again into the Wright's pipette, seal off and incubate at body temperature for 20 minutes. Then make films by the same technique as for blood-films and stain them with Jenner or Leishman stain in the usual manner. The reticulocytes present among at least 500 red cells should be counted.

### X THE COAGULATION, BLEEDING AND PROTHROMBIN TIMES

**1** The coagulation-time of whole blood *in vitro* may be determined in capillary blood by the method of Dale and Laidlaw, or more satisfactorily in venous blood by the method of Lee and White.

In the method of Dale and Laidlaw, capillary blood obtained without squeezing from a free-flowing puncture is run into a piece of capillary tubing 1·5 to 2·0 mm. in diameter and 15 mm. long with slightly contracted ends. The capillary bulb contains a small movable lead shot. It is immersed immediately in a water bath at 37°C and tilted up and down while the movement of the lead shot is observed. The coagulation time is the time which elapses between the moment of puncture and the moment when the shot ceases to move. The normal is up to 7 minutes but varies with the technique used. This method is simple but relatively insensitive.

In the method of Lee and White, venous blood is run into four un-siliconed tubes of standard size maintained at a temperature of 37°C. The coagulation time is the time from the moment of puncture to the average time at which the tubes can be tilted to an angle greater than 90° without spilling the blood. (For details of the method larger works must be consulted.) The normal coagulation time by this method is from 5 to 11 minutes (usually 6-9 minutes).

The coagulation time is increased in haemophilia, but is normal in thrombocytopenic purpura. It is also increased during the administration of anticoagulants.

**2 The bleeding time** is determined by stabbing the ear with a sharp sterile needle and blotting off the drop of blood every 30 seconds until it ceases to ooze from the puncture. The blots are recorded in series along a strip of blotting-paper and subsequently counted. The normal bleeding-time is 2 to 7 minutes. It is increased in thrombocytopenic purpura, but normal in haemophilia.

**3 The prothrombin time.** The concentration of prothrombin in the plasma is estimated by measuring the length of time taken for plasma to clot in the presence of an excess of thrombokinase and of calcium ions. Blood is withdrawn into an oxalate solution, which prevents it clotting by removing free calcium-ions, and centrifuged. To the plasma, thromboplastin—Russell viper venom or an extract of brain-tissue—is added to provide an excess of thrombokinase. Calcium chloride is then added and the time taken for the plasma to clot measured with a stop-watch. This is normally 12 to 14 seconds. Several determinations on normal subjects are made at the same time. The *prothrombin ratio* is expressed as the ratio of observed clotting time after recalcification in the specimen tested to that of normal plasma samples. The prothrombin index is the inverse of this ratio  $\times 100$ , and various arbitrary percentage scales are in use, leading to considerable confusion. The prothrombin ratio is increased in vitamin-K deficiency in the new-born, in malabsorption, in liver disease, or during the administration of anti-coagulant drugs which interfere with the synthesis of prothrombin and its co-factors.

## XI FRAGILITY OF THE RED CELLS

The fragility of the red blood-corpuscles is shown by their inability

to resist haemolysis in diminishing strengths of salt-solution. Normal salt-solution is usually taken as 0·85 per cent of NaCl, but normal red cells do not haemolyse until a dilution of about 0·4 per cent is reached. In most forms of chronic jaundice the red cells are less fragile than normal cells, and do not haemolyze in a salt solution appreciably less than 0·4 per cent. In acholuric jaundice the fragility of the red cells is greatly increased, and haemolysis takes place in strengths of salt approaching that of normal saline. Not uncommonly haemolysis begins at 0·6 per cent of salt, or even higher. The undue fragility of the red cells is the most constant and characteristic sign of this disorder, and such wide deviation from the normal is found in no other condition.

The estimation of the fragility of the red blood-corpuscles is simple. Two series of test-tubes each containing about 5 ml. of a range of dilutions of NaCl in water are put up, and to each of one series added a drop of blood from the patient under investigation and a drop of normal blood to each of the other series. It is not necessary to wash and centrifuge the red cells; the whole blood may be taken. The normal control series should never be omitted. The range of dilutions should extend from 0·8 per cent to 0·2 per cent, and there should be a difference of not less than 0·05 per cent between dilutions. The solutions can be rapidly made by placing in one burette distilled water, and in a second burette a 1 per cent solution of NaCl in water. Into the first tube run 4 ml. of the salt solution and 1 ml. of water, giving 0·8 per cent NaCl; into the following test-tube put  $\frac{1}{2}$  ml. less of salt solution and  $\frac{1}{2}$  ml. more of water, and so on until a strength of 0·2 per cent NaCl is reached. Each tube should be thoroughly shaken after the dilution is made, and again after the blood has been added. Finally, the tubes are allowed to stand at room temperature until all the intact corpuscles have settled to the bottom. The first tube showing haemolysis is recognized by the faintly pinkish colour of the supernatant fluid.

This test can be performed quantitatively, the amount of haemolysis in different strengths of saline being determined by measuring the depth of colour of the supernatant fluid by means of a colorimeter. Percentage haemolysis can then be plotted against strength of saline (Fig. 41). This shows that in normal subjects the fragility curve has an almost symmetrical sigmoid shape. Increased fragility (corresponding to increased spherocytosis) is found in familial acholuric jaundice (and only occasionally in acquired haemolytic anaemias), while decreased fragility is found in a number of anaemias, now known to be associ-

ated with the presence of abnormal haemoglobins. These are found in various parts of the world and have a particular geographical distribution. The best-known are thalassaemia (Cooley's anaemia) in persons of Mediterranean stock, and sickle-celled anaemia in persons of negroid stock. In this latter condition the red cells are normal in

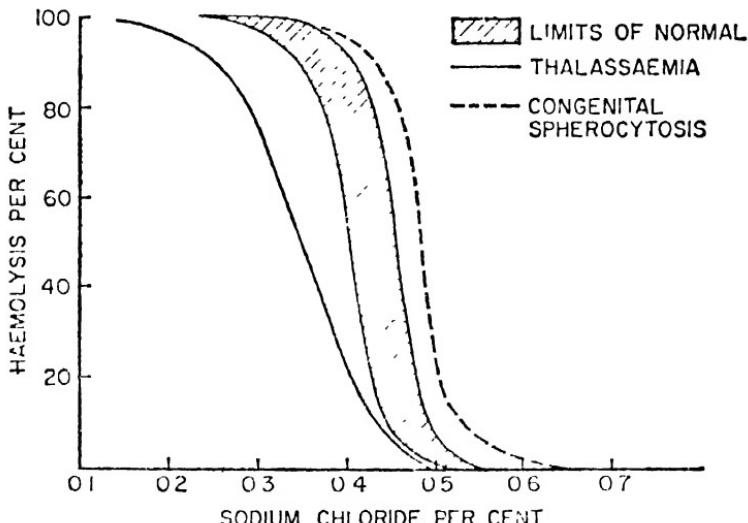


Fig. 41. Quantitative fragility curves showing increased fragility (congenital spherocytosis) and diminished fragility (thalassaemia). In general, increased fragility indicates spherocytosis and diminished fragility suggests an haemoglobinopathy.

appearance in fresh blood but 'sickle' or assume a more filamentous shape when incubated under a sealed cover-slip at a temperature of 37°C.

In summary, an increased fragility strongly suggests congenital spherocytosis (familial acholuric jaundice), while a diminished fragility suggests an anaemia associated with an abnormal haemoglobin.

## XII THE COOMBS TEST

Haemolysis in familial acholuric jaundice (congenital spherocytosis) is due to a congenital defect of the red cells. It is now known that in most cases of acquired haemolytic anaemia the haemolysis is due to the presence in the blood of antibodies directed against the patient's

own red cells. The presence of these auto-immune antibodies can be demonstrated by means of the Coombs test, which detects 'incomplete' antibodies; that is, antibodies absorbed on the surface of red cells, which are 'incomplete' in the sense that they do not cause agglutination or haemolysis when the cells are mixed with saline dilutions of the patient's own serum. The test depends on the fact that such cells, however, will agglutinate in the presence of an anti-human-globulin serum, prepared by injecting a rabbit on several occasions with normal human serum. This anti-human-globulin serum contains a globulin which draws together the 'incomplete' antibodies in the patient's red cells, and so agglutination occurs.

In the direct Coombs test the patient's red cells are thoroughly washed in normal saline at 37°C. One drop of a suspension of these cells in saline is then mixed with a drop of a potent anti-globulin serum. (At the same time a drop of the patient's red-cell suspension is mixed with a drop of saline; and normal unsensitized cells and cells known to be sensitized are also mixed with the anti-globulin serum—all as control observations.) The drops are gently rocked for 5-7 minutes and then examined for agglutination.

This test is better performed in a quantitative manner with different concentrations of anti-globulin serum, but the principle remains the same.

A positive direct test demonstrates the presence of auto-immune antibodies and suggests acquired haemolytic anaemia, but both 'false negative' and 'false positive' reactions can occur; the latter particularly in disseminated lupus erythematosus, rheumatoid arthritis, leukaemia and aplastic anaemia.

The indirect Coombs test is used to detect the presence of free auto-antibodies in the sera of patients with acquired haemolytic anaemia. The technique is complicated, but the principle is that washed normal group-O red cells are incubated at different temperatures with the patient's serum for two hours. (As a control normal serum and patient's serum inactivated by heat are also used.) If the patient's serum contains auto-antibodies against red cells, these are absorbed on to the surface of the normal red cells, which are then 'covered' with an incomplete antibody, as explained in the previous section. These red cells are then washed three times in saline and mixed with a potent anti-globulin serum as in the direct Coombs test already described. They are examined for agglutination after 5 to 7 minutes.

If antibody is detected by this test, a series of further tests can be performed to determine the characteristics and specificity of the

antibody present, but these are matters for specialists in the field.

The indirect Coombs test can also be used for the detection of Rh antibodies (p. 138) in the sera of antenatal patients.

### XIII STERNAL PUNCTURE

Sternal puncture is employed to examine the marrow in anaemias and leukaemias, to detect certain parasites, e.g. Leishman-Donovan bodies in kala-azar and in the diagnosis of carcinomatosis, myelomatosis and disorders of lipid-storage such as Gaucher's disease. For most purposes the examination of smears of marrow aspirated through a needle is quite adequate. For certain purposes the sternum needs to be trephined and a small piece of marrow excised for histological section. This is essential for an accurate diagnosis of the degree of hypoplasia present in aplastic anaemias, and to establish the diagnosis in rare cases of osteosclerosis with anaemia.

The apparatus used for puncturing the sternum is a stout trochar and cannula, fitted with an adjustable stop, and so made that a hypodermic syringe fits into the top of the cannula. The skin and tissues down to the periosteum of the sternum are anaesthetized with 2 per cent Novocain without adrenaline. A small incision is made in the skin with a tenotomy, slightly lateral to the mid-point of the manubrium or over the middle of the sternum opposite the 2nd or 3rd intercostal space. The stop of the trochar is unscrewed and adjusted according to the build of the patient; the adjustment may well be 1 cm. at first. The corticalis of the sternum is pierced with a boring motion, the stop being unscrewed to lengthen the projecting portion of the trochar if necessary. Some force may be required, but it is easy to recognize the entry of the tip of the trochar into the marrow cavity. When the cavity of the sternum has been entered, the trochar is withdrawn, the hypodermic syringe inserted, and as few drops as possible of red fluid aspirated. Without disconnecting the syringe, the cannula is removed and a swab put over the incision. The plunger of the syringe is pushed, and as quickly as possible films are made on slides from the fluid expressed from the cannula. If marrow has been successfully aspirated, the films will contain fat and appear messy and uneven. They may be stained with Jenner's or Leishman's stain.

For the interpretation of marrow films, larger works must be consulted. The normal cell count or 'myelogram' may be taken as in the table on p. 134. Photomicrographs of normoblastic and megaloblastic marrows are shown on Plate XV.

	<i>Average per cent</i>	<i>Range of normal per cent</i>
Haemocytoblasts . . . .	1 0	0 0- 2·0
Promyelocytes . . . .	4 0	0 7-14 0
Neutrophil myelocytes . . . .	13 0	2 0-20 0
Eosinophils . . . .	1 0	0 5- 3 0
Basophils . . . .	0 25	0 0- 0 5
Proleucocytes . . . .	29 0	10 0-48 0
Polymorphonuclear leucocytes . .	18 0	9 0-34 0
Total myeloid cells . . .	66 5	
Proerythroblasts . . . .	0 5	0 0- 2 0
Normoblasts, basophilic . . .	2 0	0 3- 3 0
" polychromatic . . .	12 0	5 0-15 0
" orthochromatic . .	5 0	1 0- 7 0
Total erythroid cells . . .	19 5	
Lymphocytes . . . .	10 0	2 5-24 0
Monocytes . . . .	2 0	0 0- 5 0
Plasma cells . . . .	0 5	0 0- 2 0
Megakaryocytes . . . .	6 25	0 0- 2 0
Cells in mitosis . . . .	0·25	0 1- 0 4
Smudge cells . . . .		0 3- 0 2
Myeloid-erythroid ratio . .	3 4 : 1	8 : 1 to 2 · 1

Modified from *Recent Advances in Clinical Pathology*, Churchill, London, 1947

## XV SEDIMENTATION RATE

In health, the red cells of the blood agglutinate very little on standing; the resultant clumps of corpuscles are small, and sediment slowly. On the other hand, in certain diseases the red cells agglutinate into larger clumps, which sediment more rapidly. As a sick person improves, the clumps become smaller and the sedimentation rate slower. There is no standard method for estimating the erythrocyte sedimentation rate. The Westergren method is simplest, and the one most generally in use. 0·2 ml. of 3·8 per cent sodium citrate is sucked into a 1 ml. syringe and blood is then drawn by venepuncture up to the 1 ml. mark. The mixture is shaken so that it is evenly mixed. The

blood is then sucked into a pipette 2.5 mm. in diameter and graduated to 200 mm. This is fixed in a stand and sealed by being clipped on to a cork at the bottom. The distance settled in millimetres in one hour is the sedimentation rate. The normal is 3 to 7 mm., the slightly abnormal range is 8 to 15 mm. and the grossly abnormal 15 to 110 mm. or even higher.

In the Wintrobe method a haematocrit tube (as used for determining the volume of packed cells, p. 119) is filled to the 100 mark with oxalated blood and allowed to stand vertically for one hour. By this method the normal sedimentation for men after one hour is 0 to 9 mm. and for women 0 to 20 mm. The advantage of this method is that the haematocrit tube can subsequently be centrifuged to determine the volume of packed cells, and the sedimentation rate corrected for the degree of anaemia present. This can be done by means of a chart specially prepared for the purpose (*see Amer. J. med. Sci.* (1935), 189, 102).

The sedimentation rate is reduced in polycythaemia and in congestive heart failure; and increased in anaemia, in all but the mildest toxic and infective conditions, and in many cases of cancer, especially in carcinomatosis. It is also increased in pregnancy, normal or abnormal. It rarely has any specific diagnostic value, but indicates the presence of a disease process of some sort, which may be anything from a cold to a carcinoma. It is excessively high in carcinomatosis, multiple myeloma, and disseminated lupus erythematosus. It is of some use in distinguishing between organic and functional disorders, for in the presence of a raised sedimentation rate a diagnosis of a functional disorder should never be made until all possible investigations to exclude an organic cause have been undertaken. Conversely, a low sedimentation rate does *not* necessarily exclude organic disease.

The sedimentation rate is also useful in following the progress of patients suffering from chronic diseases. It is especially useful in chronic pulmonary tuberculosis, rheumatoid arthritis and acute rheumatism. As long as the sedimentation rate is raised, there is an active disease-process, and this is frequently found a considerable time after the temperature has returned to normal. It is convenient to estimate the sedimentation rate at weekly intervals. A trend towards acceleration of the rate is an indication that the disease process is active, whereas a trend towards slowing of the rate indicates that it is subsiding.

## XV BLOOD GROUPING

Agglutination is due to the interaction of an agglutinogen (or antigen) present in red cells with an agglutinin (or antibody) present in plasma. Many different agglutinogens are present in human red cells, but in blood-transfusion the important ones are the A and B agglutinogens of Landsteiner, since these are the only ones for which the corresponding agglutinins  $\alpha$  (Anti-A) and  $\beta$  (anti-B) are normally present in human plasma. It is evident that a person's plasma cannot contain the agglutinin for an agglutinogen present in his own red cells, but with rare exceptions  $\alpha$  or  $\beta$  (or both) agglutinins are present in the plasma, whenever the red cells do not contain the corresponding agglutinogen. On the basis of the agglutinogens present in their red cells, human beings are divided into four groups—AB, A, B and O.

The agglutinogens and agglutinins present in the four groups together with the approximate percentage of individuals found in each group in England, are shown in the following table:

<i>Group</i>	<i>Corpuscles</i>		<i>Plasma</i>		<i>Percentage of individuals in groups</i>
AB	.	.	Agglutinogens AB	Agglutinins $\alpha$	3
A	.	.	" A	" $\beta$	42
B	.	.	" B	" $\alpha$	9
O	.	.	" O	" $\alpha\beta$	46

When a transfusion is given, the danger is that the agglutinins of the recipient's plasma will agglutinate the donor's red cells. How this may come about is shown in the next table, where + indicates agglutination and - no agglutination:

<i>Recipient's group and agglutinins present in plasma</i>	<i>Donor's group and agglutinogen present in red cells</i>			
	<i>AB</i>	<i>A</i>	<i>B</i>	<i>O</i>
AB Agglutinin $\alpha$	.	.	-	-
A Agglutinin $\beta$	.	+	-	+
B Agglutinin $\alpha$	.	+	+	-
O Agglutinin $\alpha\beta$	+	+	+	-

From this table it will be seen that which group any blood belongs to can be determined by means of a supply of suitable group A and B sera. The corpuscles of the blood under test will be agglutinated by both sera if it is of group AB, by group B serum only if it is of group A, by group A serum only if it is of group B, and by neither serum if it is of group O. This method of grouping is known as the indirect test. In the direct test, the donor's red cells are tested with the recipient's serum, a procedure which is often referred to as 'cross-matching'.

From this table it will also appear that blood may be given safely to any recipient from a donor of the same (or homologous) group or to any recipient from a donor of Group O (the so-called 'universal donor').

This is true in the great majority of cases, but neglects several complications, which occasionally give rise to severe and even fatal transfusion-reactions in apparently compatible transfusions.

**1 Groups A<sub>1</sub>, A<sub>2</sub>, A<sub>1</sub>B and A<sub>2</sub>B.** Group A is strictly divisible into A<sub>1</sub>, accounting for four-fifths of group A persons, and A<sub>2</sub>, accounting for one fifth. Group AB is similarly divisible into A<sub>1</sub>B and A<sub>2</sub>B. The practical importance of this fact is that A<sub>2</sub> cells react more weakly than A<sub>1</sub> cells with group B testing-sera. Hence, if a low-titre testing serum is used, group A<sub>2</sub> may be mistaken for group O and group A<sub>2</sub>B for B. This will be avoided if high-titre B serum is used, and if typing is carefully performed.

**2 Anti-O agglutinin** is present spontaneously but rarely in A<sub>1</sub>, B, and A<sub>1</sub>B sera. It reacts with all O cells and also with 95 per cent of A<sub>2</sub> cells, for which reason it is often called 'alpha<sub>2</sub> agglutinin'.

The extra agglutinins associated with these sub-groups are usually present in small amounts, unless stimulated by a previous transfusion. Hence a single transfusion of group-O blood to recipients of other groups is usually quite safe, and, in practice, reactions due to repeated transfusions are rare. One danger of repeated transfusions of O blood to recipients of other groups is, however, that the formation of anti-O agglutinins may be stimulated.

**3 The Rh factor.** If red cells from a rhesus monkey are transfused into a rabbit, the rabbit's serum will subsequently cause agglutination in 85 per cent of human bloods and no agglutination in the remaining 15 per cent: 85 per cent of human bloods are therefore described as rhesus-positive, in that they have an Rh agglutinogen in their red cells,

and 15 per cent as rhesus-negative, in that they have no such agglutinogen. Under normal circumstances Rh-negative persons have no Rh-agglutinin in their plasma, but the formation of this agglutinin may be stimulated either by a transfusion of Rh-positive blood or, in the case of Rh-negative women, by the presence of an Rh-positive foetus *in utero*, in some cases in which the father is Rh-positive. As mentioned (p. 133), the presence of Rh-antibodies in the serum can be demonstrated by the indirect Coombs test. This is an important measure in Rh-negative women, and can be used to forecast the occurrence of haemolytic anaemia of the newborn. If Rh-positive blood is given to an Rh-negative person, in whom the formation of Rh-agglutinins, has been stimulated in one of these ways, a severe or even fatal transfusion reaction may be produced. It follows that transfusion reactions in repeated transfusions may be due to this cause, rather than to the incompatibilities mentioned above, and that Rh-negative pregnant and puerperal women can only safely be transfused with Rh-negative blood of correct A, B, O group.

### Methods

**Blood-grouping.** If a transfusion is to be given, enough blood should be collected from the recipient to provide a sample of red cells for the indirect test, and a sample of plasma for cross-matching with the donor's red cells.

The recipient's finger or ear is pricked and one drop of blood allowed to drop into a small tube containing 1 ml. of a 3 per cent sodium-citrate solution. Blood from the same puncture is then allowed to run into a piece of capillary tubing about 1 mm. diameter and 2 in. long. One end of this tubing is sealed and the tube then centrifuged to separate the serum, or simply left standing, when enough serum for cross-matching purposes will usually separate out.

**Tube Method.** Into two small tubes approx. 2 in. by  $\frac{1}{4}$  in., clearly marked A and B, place respectively one volume of group-A typing-serum and one volume of group-B typing-serum, with the aid of capillary pipettes, making sure that the same pipette is never used for both sera. One volume of saline is added to each tube, and then one volume of the red-cell suspension prepared, as above. The tubes are inverted to ensure thorough mixing and then left at room-temperature for two hours. They are then gently flicked with the finger, when the presence or absence of agglutination is usually quite obvious. If there is any doubt, the contents of the tube can be taken up in a Pasteur

pipette, transferred to a slide, and examined under the  $\frac{3}{4}$  objective. Group AB cells are agglutinated by both A and B sera. Group A cells are agglutinated by group B serum, but not by group A serum. Group B cells are agglutinated by group A serum, but not by group B serum, and group O cells are not agglutinated by either group A or group B sera.

**Tile method.** This is less accurate than the tube method, but is quicker and requires less apparatus.

Large drops of group A and group B typing sera are placed on a white porcelain tile and clearly marked A and B. To each, a very small drop of the patient's whole blood is added and the tile gently rocked. Usually agglutination is clearly visible to the naked eye before the fluid dries on the tile. This method should only be used by persons with considerable experience of blood-grouping, but it is useful in an emergency.

**Cross-matching.** This must never be omitted. The simplest method is to place one volume of the recipient's serum (removed by means of a fine pipette from the capillary tube prepared as above), one volume of saline, and one volume of a 5 per cent suspension of the donor's blood in 3 per cent citrate (obtained as described under Blood-Grouping) in a small tube. The tube is then inverted, left at room temperature for two hours and examined for agglutination exactly as described under Blood-Grouping. In emergency a large drop of the patient's serum may be mixed on a slide with a drop of a 5 per cent suspension of the donor's red cells obtained as above. The slide should then be left for ten minutes with occasional rocking and examined under the  $\frac{3}{4}$  objective.

More satisfactory cross-matching may be achieved by carrying out the Coombs test, using the donor cells and the recipient serum. Many modifications have been devised to render cross-matching more sensitive, and special reference books should be consulted. Rare antigenic blood-groups, other than the ABO and Rh systems, may give rise to difficulty; but a carefully conducted cross-match of cells as above, should avoid serious transfusion-reactions, and if repeated incompatibility is found on the cross-match, assistance from a specialist centre should be sought.

## XVI THE 'LE' CELL PHENOMENON

If anticoagulated or defibrinated blood from a patient with disseminated lupus erythematosus is incubated *in vitro* for two hours,

the nuclei of some polymorphonuclear leucocytes are lysed and taken up by other polymorphonuclear leucocytes. The nuclei appear as large spherical bodies staining pale purple in the cytoplasm of the ingesting cell, which is then known as an LE cell. Such cells are not found in fresh blood or bone marrow from patients with DLE, but only appear after incubation *in vitro*. If repeated examinations are made, they are found in a high proportion of patients suffering from disseminated lupus erythematosus, and they are not found in patients suffering from other diseases.

Two methods are in general use. The patient's defibrinated blood may be incubated for 2 hours at 37°C. The blood is then centrifuged, and films made from the buffy coat are stained with Jenner's or Leishman's stain. Alternatively the patient's serum is incubated with a suspension of normal leucocytes, when the same phenomenon may be observed.

## XVII PARASITES IN THE BLOOD

These may be looked for in fresh blood under a coverslip, in which some of them, e.g. microfilariae and trypanosomes, may be seen alive and moving; or in fixed and stained films, which may be thick or thin. The making of thin films has already been described (p. 112).

**Fresh films.** The slides and cover-slips must be clean and free from grease, and should be held by the edges so that the fingers do not touch their surfaces. Prick the finger or lobe of the ear and wipe away the first drop of blood. When a second has accumulated, just touch it with the centre of a cover-slip and lower the latter carefully on to the centre of a slide. The blood spreads out into a film. If a drop of blood of the right size has been used the centre of this film is almost colourless. If the centre is red, apply a little pressure. The film should be examined, with the light well cut down, in turn under the  $\frac{3}{4}$ ,  $\frac{1}{2}$ , and, if necessary,  $\frac{1}{4}$  objectives.

**Thick films.** The thick-film method enables a much larger amount of blood to be examined in a given time than is possible with thin films, and is therefore valuable for the detection of parasites when these are present in small numbers. Take a drop of blood on the centre of a slide, and spread it with a triangular needle until print can be clearly seen through it while it is still wet. Allow the film to dry thoroughly,

by leaving it protected from dust for a minimum of two hours and if possible overnight. If speed is essential, it may be dried by warming very cautiously. When the film is thoroughly dry it must be dehaemoglobinized by immersing the slide gently in an upright position in a beaker of clean and preferably distilled water, and allowing the haemoglobin to dissolve out. Gentle movement helps, but if the slide is handled roughly the film may become detached. In about five minutes, when the film is colourless and opaque, it is removed from the water and allowed to dry. Thereafter it can be stained with Jenner's or Leishman's stain, in the same manner as a thin film.

Another method uses a dilute watery solution of Giemsa's stain, which dehaemoglobinizes and stains the film in one operation. The slide with the film facing downwards is placed across two thin glass rods in a flat glass dish and a freshly prepared dilute watery solution of Giemsa's stain (1 drop of stain to each ml. of water) is run under the slide and left for 15 to 20 minutes. The slide is then washed gently in a dish of water and allowed to dry in a semi-vertical position. Thick films should be inspected rapidly under the  $\frac{3}{4}$ -in. objective and then examined systematically under the oil immersion.

**Recognition of parasites.** The important parasites of the blood are the parasites of malaria, microfilariae of several varieties, trypanosomes, Leishman-Donovan bodies and the spirochaetes of relapsing fever.

For the diagnosis of *malaria*, thick films should be used for the detection of parasites and thin films for their identification. Films should preferably be taken when the patient's temperature is raised. Thick films stained as described should be examined systematically for 10 to 15 minutes before concluding that no parasites are present. The recognition of parasites in thick films requires practice. White cells, platelets, bacteria, the remains of reticulocytes, and miscellaneous dirt can be mistaken for parasites. Parasites have definite morphological and staining properties, and objects which do not show these are not parasites.

For details of the identification of the different types of parasites in thin films, larger works must be consulted. The main distinguishing points are as follows (Plate XIV). In infection with *Plasmodium falciparum*, which produces malignant tertian malaria, schizogony generally takes place in the tissues, so that except in rare cases in moribund patients, ring forms and a few crescent-shaped gametocytes only are seen in the peripheral red-blood cells. Ring forms of any

species consist of a rim of cytoplasm which stains blue and a small nucleus or chromatin dot which stains red. The rings of *Plasmodium falciparum* are usually, though not invariably, small and delicate, and the red cells are not enlarged. More than one ring may appear in a single red cell, and some rings may have two chromatin dots. Marginal forms or 'formes appliquées' with the parasite lying along the edge of the cell may be seen. A few crescent-shaped gametocytes, which are easily recognized (Plate XIV: 13 and 14), may be seen in films from untreated patients, but if absent at this time they may appear, particularly in patients treated with mepacrine, some seven to ten days after the beginning of treatment.

In the remaining three species, schizogony takes place in the peripheral blood, so that ring forms, large trophozoites, and schizonts will be present together in films.

In infections with *Plasmodium vivax*, which produces benign tertian malaria, the rings are large and stout and often measure one-third of the diameter of the red cell. The red cells may be enlarged, and if properly stained may show well marked Schüffner's dots (Plate XIV: 1 and 2). Large irregular trophozoites containing brown pigment, and mature schizonts with 16 or more merozoites, may be seen (Plate XIV: 3).

In infections with *Plasmodium malariae*, which produces quartan malaria, the ring forms are also large and stout, but the larger trophozoites are more compact and dense-looking and frequently take a characteristic band form (Plate XIV: 6 and 7). Mature schizonts contain some eight merozoites arranged in a rosette form around a mass of pigment. Further, Schüffner's dots are not seen and the red cells are not enlarged.

In infection with *Plasmodium ovale*, which is much the rarest of the four species, the parasites have some of the characteristics of plasmodium vivax (e.g. large and prominent Schüffner's dots) and others of plasmodium malariae (e.g. compact large trophozoites and occasional band forms). The most characteristic feature is the distortion in shape of the red cells, which become oval or fimbriated, and the schizonts only contain 8 to 10 merozoites (Plate XIV: 9 and 10).

In some cases, mixed infections may be present.

In the diagnosis of *trypanosomiasis*, examination of the blood is generally less efficient than the examination of fluid obtained by gland puncture. An enlarged gland, usually in the posterior triangle of the neck, is held firmly between the thumb and fingers of the left hand, while a moderate sized hypodermic needle is plunged through

the skin and into the substance of the gland. A small amount of gland-fluid passes into the needle, and suction is neither necessary nor desirable. The needle is withdrawn and its contents blown out on to clean glass slides. The fluid should be examined fresh and unstained, as described for fresh blood films, and thin films should be stained with Leishman's stain.

Trypanosomes (Plate XIV: 20) may also be found in thick or thin blood films and, in advanced cases, in films made from the deposit of centrifuged cerebro-spinal fluid. (For methods of concentrating trypanosomes in the blood, larger works must be consulted.)

The important trypanosomes of man are *Trypanosomata gambiense* and *rhodesiense*, which cause African sleeping sickness, and, as seen in human blood, are usually morphologically indistinguishable; and *Trypanosoma cruzi*, which causes Brazilian trypanosomiasis. The latter exists chiefly in a non-flagellated form in the organs and muscles, and only occasionally appears in the blood as a flagellate trypanosome (Plate XIV: 17).

In a suspected case of trypanosomiasis, several specimens of gland-fluid and of blood should be examined, both fresh and unstained, and in stained films. Fresh films should be examined under the  $\frac{3}{4}$  and then under the  $\frac{1}{2}$  objective. Under the latter trypanosomes may be seen 'lashing' their way amongst the cells, and are often first detected by the commotion they produce in the latter. This movement must not be confused with that produced by the *Treponema* of relapsing-fever (Plate XIV: 18).

In stained films, examined under the oil-immersion lens, typical trypanosomes are seen as elongated fusiform structures some 14 to  $30\ \mu$  long and 1 to  $3\ \mu$  broad with a longitudinal undulating membrane and a terminal flagellum projecting from the anterior end (Plate XIV: 17). There is a centrally placed nucleus, and at the posterior end a smaller black-staining kinetoplast. The shorter forms are more stumpy and may have little or no free flagellum.

In the diagnosis of *kala-azar*, Leishman-Donovan bodies may be looked for in the blood or in material obtained by sternal, gland, spleen, or liver puncture. Of these, examination of marrow obtained by sternal puncture (p. 133) is probably the simplest and safest method, but in occasional cases the parasites may be found by the examination of stained blood films, when they are seen in the cytoplasm of large mononuclear cells. When direct microscopic examination fails, culture methods (for which larger works must be consulted) are frequently successful.

## 144 CLINICAL EXAMINATION OF THE BLOOD

The bodies may be seen in thick or thin stained blood films which should be searched systematically under the oil-immersion objective. The parasites are seen as round or oval bodies from 2 to 5  $\mu$  in diameter, containing a large round or oval solid-looking nucleus and a smaller more deeply stained and usually rod-shaped kinetoplast. In Leishman-stained films the cytoplasm is blue and the nucleus and kinetoplast are red (Plate XIV: 16).

While the term Leishman-Donovan bodies strictly applies to the Indian form of kala-azar, exactly similar Leishmania may be found in the blood or tissues in cases of Mediterranean kala-azar; from fluid obtained by puncture at the margin of the lesion in the various forms of cutaneous leishmaniasis or 'tropical sore', and from the mucous membranes of the mouth, nose, or throat in espundia or South American leishmaniasis.

Adult filarial worms or macrofilariae are parasites of the lymphatic system or connective tissues. Their presence is diagnosed by the finding of their larvae or microfilariae in the blood-stream. Three main varieties of microfilariae are found in the blood of man. These are: (1) *Filaria bancrofti*, which is found in the blood-stream in any numbers only at night, and which causes filariasis, characterized by irregular fever, lymphangitis, and various forms of elephantiasis; (2) *Filaria loa-loa*, which is found in the blood-stream only by day, and which causes loaisis, characterized by transient red painful swellings known as Calabar swellings; and (3) *Filaria perstans*, which is non-periodic, appearing equally by day and night, and has no recognized pathogenic effects.

If filariasis is suspected, blood should be examined say at 8 a.m., noon, and 4 p.m., and again at 8 p.m., midnight, and 4 a.m. Fresh unstained films should be used for the detection of the filariae, and stained ones, thick if the larvae are scanty and thin if they are plentiful, for their identification.

In fresh unstained films microfilariae are easily seen under the  $\frac{1}{3}$  objective, as actively-moving linear objects. In stained films they are seen as wormlike objects with a round head and a pointed tail, from 5 to 8  $\mu$  broad (i.e. about the diameter of a red cell) and from 100 to 300  $\mu$  long (Plate XIV: 19). The main differentiating features of the three species, apart from their periodicity, are as follows. *Filaria bancrofti* and *F. loa-loa* have a delicate sheath, which can be seen where it projects beyond the rounded head and pointed tail of the larva, whereas *F. perstans* is unsheathed. All larvae have a central column of nuclei extending from the head more or less to the tail. In

*F. loa-loa* and *F. perstans* the nuclei extend to the extreme tip of the tail, whereas in *F. bancrofti* the column ends short of the tip.

Microfilariae do not stain well by Leishman's stain, and a better staining method is as follows. Thick films should be dehaemoglobinized, as described under malaria, and allowed to dry. Thereafter the method is the same for thick or thin ones. The films are fixed for 5 minutes with methyl alcohol and rinsed in water. The slides are then flooded with haematoxylin—preferably Ehrlich's mixture, though Mayer's haemalum can also be used—which is gently warmed until steam rises and an occasional bubble is seen. The heat is withdrawn for a few minutes and then reapplied, the process being repeated several times in a period of 15 minutes. If the slide shows signs of drying, additional stain must be added. When cool, the slide is immersed in water to wash off the stain and then allowed to stand in running tap water until a blue colour appears, generally in about 15 minutes. When quite dry, it should be mounted in Canada balsam. Microfilariae are readily recognized by the intense staining of their nuclei, and the sheath, if present, is easily seen.

The *Treponema recurrentis* of relapsing fever should be sought under the  $\frac{1}{2}$  objective in fresh unstained films of blood and in thin films stained by Leishman's method under the oil-immersion.

Fresh unstained films should be examined with the light well cut down. Agitation of the red cells usually calls attention to the presence of parasites, which may otherwise be difficult to detect.

In thin stained films the spirochaete is seen as a linear object with tapering ends,  $0.4 \mu$  in breadth and 10 to  $30 \mu$  in length. The spiral shape which it possesses in life is lost and the body lies in irregular curves. In searching for the organism, it is important to direct the eyes deliberately to the spaces between the red cells rather than to the red cells, or the parasite may be missed (Plate XIV: 18).

# **6 THE RESPIRATORY SYSTEM**

## **I ANATOMICAL LANDMARKS**

**Lobes of the lungs.** It is important to know the limits of the individual lobes of the lungs. A line from the 2nd thoracic spine to the 6th rib in the mammary line corresponds to the upper border of the lower lobe (the major interlobar fissure). A horizontal line on the right side from the sternum at the level of the 4th costal cartilage, drawn to meet the first, marks the boundary between the upper and middle lobes (the minor interlobar fissure). The greater part of each lung, as seen from behind, is composed of the lower lobe, only the apex belonging to the upper lobe; while the middle and upper lobes on the right side, and the upper lobe on the left, occupy most of the area in front. In the axillary regions, parts of all the lobes are accessible.

**The bifurcation of the trachea** corresponds in front with the lower border of the manubrium sterni, that is with the angle of Louis; behind, with the disc between the 4th and 5th thoracic vertebrae.

The 12th rib cannot always be felt and so it is not wise to count the ribs from below upwards. These are best counted downwards from the second costal cartilage. This cartilage articulates with the sternum at the extremities of the angle of Louis, a transverse bony ridge at the junction of the body and the manubrium, which is easily felt beneath the skin.

**Anatomy of the bronchi.** The two main bronchi each give off four main branches: on the right, one to the upper lobe, one to the middle lobe, one to the dorsal lobe (the upper and posterior part of the lower lobe), and one to the remainder of the lower lobe; and on the left, one to the upper lobe proper, one to the lingular process of the upper lobe (which represents the middle lobe on the left side), and a dorsal and lower lobe bronchus as on the right side. These main bronchi then divide into segmental bronchi, which supply individual

segments of lung. It is important to have a working knowledge of these segments, because it is often possible from the signs and X-ray appearance present to determine which segment and which segmental bronchus is affected by disease. The accompanying diagrams (Figs. 42-44), give a simplified scheme of present knowledge of the anatomy of the segmental bronchi, and indicate the 'respiratory districts' or bronchopulmonary segments supplied by them.

## II INSPECTION

### A. Form of the Chest

The shape of the chest varies with the build of the individual, often being short, broad and deep in the thick-set, and long, flat and narrow in the tall and spare. There is also wide variation in the thickness of the muscles and the slope of the shoulders in healthy people. Estimates of the significance of variations in the shape of the chest must, therefore, take into account the build of the individual. Normally the chest is bilaterally symmetrical, and on cross-section is elliptical. Its shape is affected by extra-pulmonary as well as pulmonary disease. For example, in the past, rickets was a common cause of chest deformity, the combination of softness of the bones and obstruction to respiration (due to adenoids and chronic or recurrent upper respiratory tract infection) leading to the deformities known as pigeon-chest and Harrison's sulcus.

Disease of the vertebral column can produce *kyphosis* (forward bending) or *scoliosis* (lateral bending), which often occur together. These lead to obvious asymmetry of the chest, and may decrease the size of the thoracic cage and restrict lung-movement. Scoliosis may also lead to clinical and radiological displacement of the trachea and apex-beat. The spine and rib-cage may become particularly immobile in ankylosing spondylitis, which may lead to a fixed kyphosis.

*Pulmonary disease* produces several important deformities of the chest. Unilateral apical fibrosis due to tuberculosis may cause obvious flattening of one apex, whilst more extensive unilateral fibrosis or collapse in childhood can even lead to scoliosis. Obstructive airways disease (p. 168) leads to a *barrel-shaped chest*. There is obstruction to expiration, and the lungs become over-inflated, so that the chest becomes fixed in what is normally the position of full inspiration. Since no further expansion is possible, the rib-cage tends to move up in one piece by the action of the accessory muscles of respiration,

*The Right Lung*

Upper lobe	Apical bronchus and segment
	Posterior bronchus and segment
	Anterior bronchus and segment
Middle lobe	Lateral bronchus and segment
	Medial bronchus and segment
Lower lobe	Apical bronchus and segment
	Medial basal bronchus and segment (bronchus not shown on diagram)
	Anterior basal bronchus and segment
	Lateral basal bronchus and segment
	Posterior basal bronchus and segment

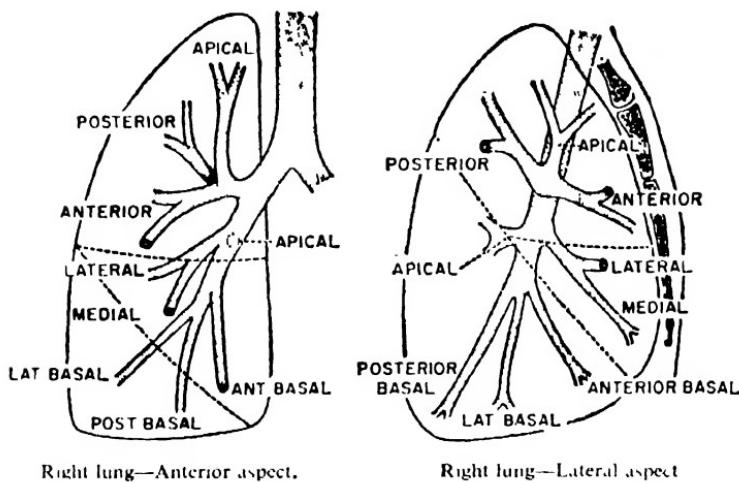


Fig. 42. Right lung showing diagram of segmental bronchi.

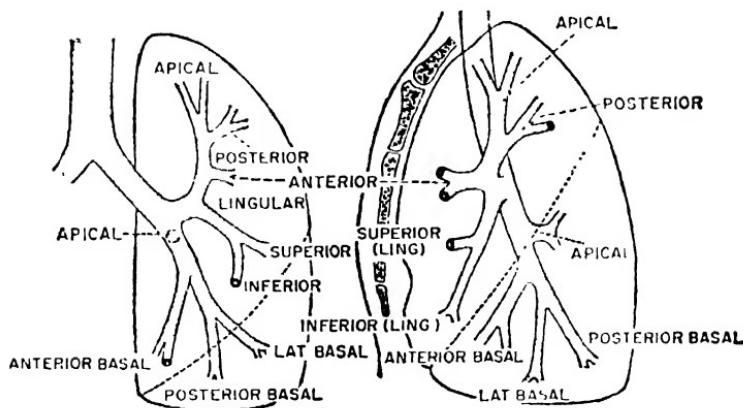
which become more prominent than usual. The ribs are less obliquely set than usual, the anterior-posterior diameter of the chest is increased, the spine becomes unduly concave forwards, and the sternum is more arched than usual, with a prominent angle of Louis.

### B. Movements of the Chest

The *rate of respiration*, for a normal adult at least, is about 14 to 18 per minute, but there is a wide margin on either side of these figures. An increase rate of respiration or *tachypnoea* is an important sign of pulmonary disease, which is frequently missed owing to lack of observation or inaccurate counting. Increased rate may result from exertion, nervous excitement, fever, or anoxaemia, which may be due

*The Left Lung*

Upper lobe	Upper division bronchus	apical bronchus and segment posterior bronchus and segment anterior bronchus and segment
	Lingular (lower division) bronchus	superior bronchus and segment inferior bronchus and segment
Lower lobe	Apical bronchus and segment	
	Anterior basal bronchus and segment	
	Lateral basal bronchus and segment	
	Posterior basal bronchus and segment	



Left lung—Anterior aspect.

Left lung—Lateral aspect.

Fig. 43. Left lung showing diagram of segmental bronchi.

to cardiac, pulmonary, bronchial or laryngeal causes, to alteration in the oxygen-carrying power of the blood, or to interference with the normal reflex control of respiration by structural changes in the lungs. It may also arise from the association of pain with breathing, as in pleurisy and peritonitis, when the breathing becomes shallow and must therefore be more frequent to make up for the slighter expansion.

The *ratio between respiration and the pulse* in health is about 1 to 4. In severe pneumonia respiration may occur almost as frequently as the pulse. In certain cases of narcotic poisoning, the ratio may become 1 to 6 or 7.

The *rhythm* varies very considerably even in health, and if the act is performed consciously it may become very irregular. Study it, therefore, when the patient is off his guard, as only then can accurate

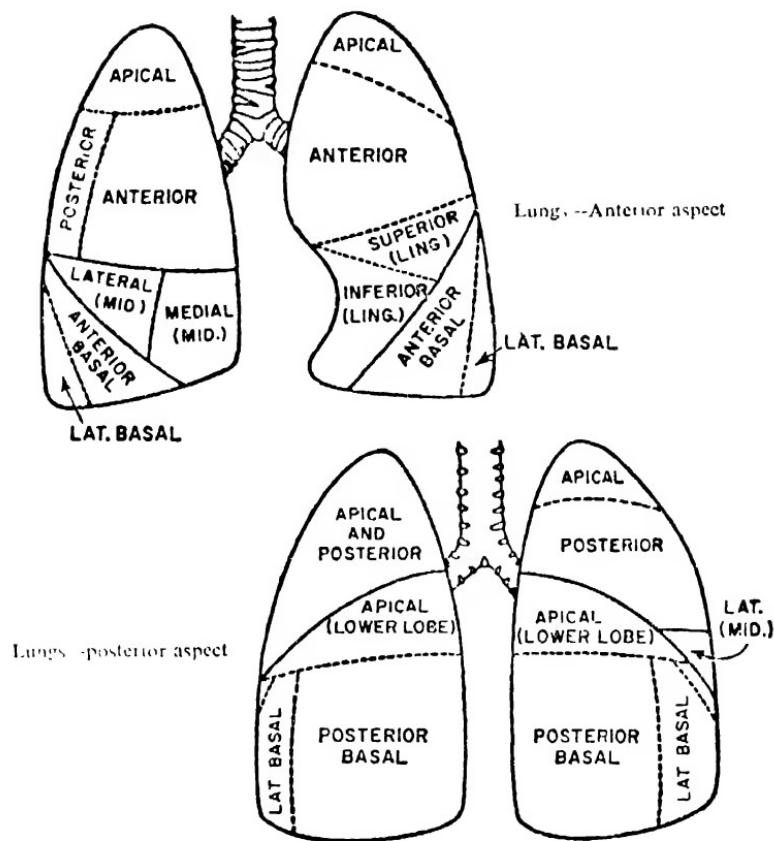


Fig. 44. Diagram of 'respiratory districts' supplied by segmental bronchi.

(*Ligs. 42-4 are reproduced with the kind permission of Dr Robert Coope and E. & S. Livingstone Ltd from Keers and Rigden, 'Pulmonary Tuberculosis'.*)

observations be made. Either inspiration or expiration may be unduly prolonged, the former being commonly associated with laryngeal or tracheal diseases, the latter with bronchial or pulmonary. A peculiar type, where successive respirations gradually get deeper and deeper

till a maximum is attained, and then fall off again until a pause of complete apnoea occurs, to be followed by another wave of gradually deepening and then diminishing respiration, is known as *Cheyne-Stokes breathing*. The pause may last for fully half a minute, though it is often shorter, and the whole cycle is usually completed in less than two minutes. It is most conspicuous when the patient who exhibits it is asleep or unconscious, but may be overlooked if the patient is awake, and particularly if he is talking. Cheyne-Stokes breathing occurs most commonly in cardiac and renal failure, severe pneumonia, increased intracranial pressure and narcotic drug poisoning.

*Movement.* Note its amount, whether it is expansile in character, and whether it is similar, or different, on the two sides and over corresponding areas. Amount of movement and expansion are by no means interchangeable terms; in emphysema the chest may move considerably, but there is little expansion. Chest expansion in men can be measured with a tape measure round the chest just below the nipples. In a fit young man it may be 2-3 inches; in emphysema it may be  $\frac{1}{2}$  in. or less.

Diminished or absent expansion may be due to pleurisy with effusion, pneumothorax, consolidation, collapse, fibrosis, or the presence of a neoplasm. It is also found in tuberculosis and lobar pneumonia, the former especially at the apices, the latter at the apex or base according to the situation of the disease.

### III PALPATION

Before making a systematic examination, it is well to lay the hand on any part of the chest which presents an obvious swelling, or where the patient complains of pain. Look at the face rather than the part under examination, so as to avoid causing unnecessary pain. Pain may be due to recent injury to the chest-wall, or to inflammatory conditions; to intercostal muscular pain, where, as a rule, specially painful spots can be discovered on pressure; to a painful costochondral junction; to secondary malignant deposits in the ribs; to herpes zoster, before the appearance of the eruption; or to pleurisy. In the case of pleurisy, pressure may considerably increase the pain. Pain may also be due to cardiac causes, such as coronary thrombosis and pericarditis. At the same time the nature of any swelling should be investigated. Fluctuation occurs when an abscess has formed in the chest-wall.

The positions of cardiac impulse and trachea should then be

determined. Feel for the trachea in the suprasternal notch, and decide whether it is placed centrally, or deviated to one or other side, by its relation to the suprasternal notch and the insertion of the sternomastoids. Slight deviation of the trachea to the right may be found in healthy people. Displacement of the cardiac impulse alone may be due to scoliosis, the commoner form with its convexity to the right causing a displacement of the cardiac impulse to the left and vice versa, to funnel depression of the sternum or to enlargement of the left ventricle, particularly in aortic incompetence and hypertension. In the absence of these conditions, a significant displacement of the cardiac impulse or trachea, or of both together, suggests that the position of the mediastinum has been altered by disease of the lungs or pleura. The main conditions which 'push' the mediastinum away from the affected side are pleurisy with effusion and pneumothorax, and the main conditions which draw it towards the affected side are fibrosis of the lung in tuberculosis or after broncho-pneumonia, and collapse of one or more lobes. In fibrosis or collapse of the upper lobe of a lung, the trachea only is displaced.

The *nature of the respiratory movements* must next be studied. It is important to make certain that the two sides of the chest move to approximately the same extent. This is done by fixing the finger-tips of either hand at the patient's sides, and making the tips of the thumbs just meet in the middle line in front of the chest. The patient is directed to take a full inspiration, when the distance of departure of the thumbs from the middle line indicates the extent of expansion of either half of the chest. The causes of diminished expansion have been mentioned under inspection.

*Vibrations* may be detected by palpation. For this purpose the palm of the hand should be applied flat on the chest. The patient is then told to repeat 'one, one, one', or 'ninety-nine', in a clear voice. The hand placed on the thorax detects distinct vibration whilst this is done, and one must determine whether the vibrations in corresponding areas on the two sides of the chest are approximately equal in intensity—not, however, forgetting that where the heart encroaches on the left lung the fremitus is necessarily much diminished. *Vocal fremitus is increased* when the lung is consolidated, as in lobar pneumonia, or contains a large cavity near the surface. *Vocal fremitus is diminished* when the corresponding bronchi are obstructed, or totally absent when the lung is separated from the chest-wall by a pleural effusion. The cause in this case is not that fluid is a bad conductor of sound or of vibration—the reverse is the case—but that

the collapsed lung itself fails to convey the vocal fremitus, and so the vibrations never reach the fluid. In young persons and women, the vocal resonance is different both in character and intensity from that in male adults.

#### IV PERCUSSION

**Method of percussion.** The middle finger of the left hand is placed *firmly* on the part which is to be percussed, and is adapted to any inequalities of surface, so that no air-space is interposed between it and the skin. The back of its middle phalanx is then struck with the tip of the middle finger of the right hand. The stroke should be delivered from the wrist and finger-joints, not from the elbow, and the percussing finger should be so bent that when the blow is delivered its terminal phalanx is at right angles to the metacarpal bones, and strikes the pleximeter finger perpendicularly. As soon as the blow has been given, the striking finger must be raised, lest it should impair the vibrations it has excited, just as the hammers of a piano fall back from the wires as soon as these have been struck. The blow should be no heavier than is necessary to elicit the resonance of the part being examined, and the wrist-joint must move loosely. Good percussion is a knack, which requires much practice.

*Three rules* should always be remembered when percussion is being carried out. In defining the boundaries between contiguous organs, the percussion should invariably be performed from the resonant towards the less resonant. The longer axis of the pleximeter finger should be parallel to the edge of the organ whose delimitation is being attempted, and the line of percussion should be at right angles to that edge. The pleximeter finger must be kept in firm contact with the chest-wall. It is seldom necessary to deliver more than two or three strokes at any one situation; repeated blows cause much discomfort to a sensitive patient.

The *character of the sound produced* varies quantitatively and qualitatively. When the air in a cavity of sufficient size and appropriate shape is set into vibrations which are not modified by excessive tension of the containing walls of the space, the sound heard has a tympanitic character; such a note is heard on percussion over an air-containing viscus, such as the stomach; but when the cavity is subdivided into a number of small loculi by numerous septa, more or less tense, a characteristic resonance, no longer tympanitic, is produced. Such conditions prevail in the healthy lung, and the observer must learn by

practice to recognize its distinctive quality. In general terms, this pulmonary resonance is low in pitch and clear in character.

Beginning in front, the examiner should tap lightly and directly (i.e. without pleximeter finger) on the most prominent point of each clavicle —being sure that the points examined correspond exactly with each other—and should observe the quality of the sound, and particularly whether the effects on the two sides are identical. Thereafter the other corresponding areas on either side should be carefully percussed in the manner already described. The presence of the heart will interfere, in parts of the left side, with the development of a sound resembling that from the corresponding point on the right. The back and axillary regions should then be examined in the same manner.

It is essential in all parts of the examination that the patient's attitude is a comfortable one, and that his arms and shoulders are placed symmetrically, whether he is sitting up or lying down. When in the case of a very ill patient it is only possible to examine the back by rolling him on to his side, only gross differences in the note on the two sides are significant. If possible the patient should be examined lying first on one side and then on the other. Should the patient's chest be asymmetrical, from scoliosis or other cause, equal resonance on the two sides is not to be expected, and again only gross differences between the two sides are significant:

The observer should have two objects in mind: first, to make a comparison of the percussion note in comparable areas on the two sides; and second, to map out the limits of lung resonance particularly at the apices, the bases, and the area of cardiac dullness.

The *normal degree of resonance* varies from individual to individual and in different parts of the chest in the same individual, being most resonant below the clavicles and scapulae where the muscles are relatively thin, and least resonant over the scapulae.

The lower limits of lung resonance should be determined by percussing from above downwards. The *lower border of the right lung* lies over the liver, and is thin; therefore its exact situation is best made out by light percussion. Posteriorly, however, the muffling due to the thick muscles and fat of the back makes it necessary to percuss more firmly. When the patient is obese, very heavy percussion with several fingers may be necessary. In quiet respiration and on light percussion the lower border is found to lie in the mammary line at the 6th rib, in the midaxillary line at the 8th rib, in the scapular line at the 10th rib. On heavier percussion some loss of resonance, due to the underlying

liver and diaphragm, is found at higher levels and in the mammary line can be detected from the 4th interspace downwards.

*On the left side* the lower border overlaps the stomach, and so the transition is not from lung-resonance to dullness, but to tympanitic stomach-resonance. Posteriorly, however, the splenic dullness and the dullness of the various solid structures which lie below the lung near the spine are interposed, so that the conditions resemble those found on the right. The position of the lower border corresponds pretty closely with that on the right side; it may, however, be found a trifle farther down.

In disease the resonance may be increased or decreased.

*Resonance is increased* when the pleural cavity contains air and the lung is more or less collapsed towards the hilum. The note varies from one that is hyper-resonant to one that is distinctly tympanitic, according to the amount of air in the pleural cavity.

A characteristic form of high-pitched tympanitic resonance, the 'bruit d'airain' (airain = brass), 'bell-sound', or 'coin-sound' may also sometimes be heard in pneumothorax, by percussion over the front of the chest with a couple of coins—one being used as a plexor and the other as a pleximeter—whilst the observer listens with the stethoscope at the back of the patient. Failure to elicit the 'bruit d'airain' does not mean, however, that a pneumothorax is not present.

Though a definite tympanic of hyper-resonant sound is regularly found in pneumothorax, it is not consistently found in any other pulmonary conditions. It may be found over some large cavities. It is often stated that resonance is increased in emphysema. When, however, a group of doctors at the Royal Postgraduate Medical School were asked to percuss the chests of patients with emphysema and those without chest disease, without knowing the diagnosis, several normal chests were described as being hyper-resonant. In the diagnosis of emphysema it is more important to note the increased limits of resonance with loss of liver and cardiac dullness than the quality of the resonance.

*Resonance is diminished* when the pleura is thickened, when the underlying lung is more solid than usual for any reason and when the pleural cavity contains fluid. Thus, there may be slight impairment of resonance at one apex in a case of pulmonary tuberculosis due to local infiltration and fibrosis. Considerable impairment may be found over areas of lung affected by fibrosis or collapse. In lobar pneumonia, percussion over a completely consolidated lobe produces a definitely dull note, whilst absolute dullness along with a peculiar sensation of

resistance in the percussing finger, so-called 'stony dullness', is the characteristic finding over a pleural effusion of any size. In heart-failure, impaired resonance or dullness may be found at the bases of both lungs, indicating oedema of the bases or bilateral effusions.

**Myotatic irritability.** In certain conditions the muscles on the front of the thorax are unduly irritable, and a light tap over the sternum produces contractions, at some distance off, in the pectoral muscles. This phenomenon occurs in any wasting disease, and is known as myotatic irritability or myoedema.

## V AUSCULTATION

Three observations must be made at each point auscultated: first, the character of the breath-sounds; second, the character of the vocal resonance; and third, the presence or absence of other sounds.

For good auscultation, a patient in bed should lean back against pillows, or lie on his back, and be completely relaxed. To examine the back, the patient should sit up, but if he is unable to do this he should be rolled round first to one side then to the other. In serious cases, the minimum necessary examination should be done. Take care that the chest-piece is accurately applied, that it is not allowed to move on the surface of the skin, and that no undue pressure is exerted. The patient should breathe with his mouth open, regularly and fairly deeply, but not noisily. It is quite useless to attempt auscultation of a patient who is shivering.

### A. Character of Respiratory Sounds

There are two typical varieties of breath-sounds, both of which are audible in health at certain parts of the chest, and these must be carefully studied. The first is known as vesicular breathing, the second as bronchial. Vesicular breath-sounds are produced by the passage of air in and out of normal lung-tissue, and are heard all over the chest under normal conditions. Bronchial breath-sounds are produced by the passage of air through the trachea and large bronchi. Under normal conditions they can be heard by listening over the trachea, but they are not heard over normal lung-tissue (except where they may modify the sounds heard over normal tissue situated near the trachea and large bronchi, as will be mentioned later). In disease, however, these sounds may be conducted from the bronchi to the chest-wall, as, for instance, when a whole lobe is consolidated by pneumonia. Under these circumstances no air enters or leaves the alveoli and no

vesicular breath-sounds can be heard. Provided, however, that the bronchi are patent, so that bronchial sounds are produced and conducted through them, and provided that sufficient lung is consolidated to convey these sounds to the chest-wall, bronchial breathing will be heard over the area affected by these changes.

In *vesicular breathing*, which can be heard typically in the axillary and infrascapular regions of a healthy individual, the following facts pertain:

The inspiratory sound is fairly intense, and is audible during the whole of the act. The pitch is low, and the quality is characteristic, being somewhat rustling. The expiratory sound follows that of inspiration without a distinct pause. It only remains audible during the earlier part of the expiratory phase, and under normal conditions the inspiratory sound is heard for at least twice as long as the expiratory.

To learn to recognize *bronchial breathing* the student should listen over the trachea, though he must not expect to hear so intense a type of bronchial respiration when he subsequently examines a diseased lung. The inspiratory sound is moderately intense. It becomes inaudible shortly before the end of inspiration. Its quality is harsh and aspirate. The expiratory sound is generally more intense than the inspiratory; the pitch is often higher; the duration extends through the greater part of expiration, being as long as, or even longer than, the inspiratory sound. In quality it exactly resembles the inspiratory sound, being aspirate in character. Bronchial breathing can most readily be recognized by the quality of the expiratory sound and the definite gap between inspiration and expiration.

**Vesicular breathing.** The principal variations which can be detected in vesicular breathing are as follows:

- i. *Puerile.* The sounds are harsher than in the adult, but have a similar duration.
- ii. *The breath-sounds may be reduced in intensity or absent,* and this may affect the whole chest or be confined to one or more areas. In persons breathing quietly in health the breath-sounds may be almost inaudible. This has no pathological significance, and the sounds become audible if the subject is asked to breathe more deeply with the mouth open.

Local diminution or absence of the breath-sounds in one or more areas may, however, be an important sign of disease. It is found in

the presence of greatly thickened pleura, pleural effusions, and pneumothorax. It is also found in any condition causing a diminished air entry to the underlying lung, when the conditions necessary for the appearance of bronchial breath-sounds are not present. It may be found, for example, in pulmonary tuberculosis—usually at one apex; in lobar pneumonia, before consolidation is established—usually at one base; in heart failure—usually at both bases; or over areas of lung affected by fibrosis or collapse from any cause.

Over a *pleural effusion*, breath-sounds of any kind are absent as a rule, probably because, though fluid itself is a good conductor of sound, a combination of partly-collapsed lung and fluid does not form a sufficiently uniform conducting medium. Occasionally it happens that when a considerable quantity of fluid has accumulated, the breath-sounds, instead of disappearing, become loud, and possess a marked bronchial character. In such cases the vocal resonance also is loud, but is usually more or less aegophonic. This exceptional state is most commonly observed posteriorly over the lower lobe of the lung in children.

Some prolongation of the expiratory sound is characteristic of asthma and emphysema, and this must not be mistaken for bronchial breathing. It is due to the fact that in these diseases the act of expiration is itself performed more slowly than in health.

## 2. Bronchial breathing may be noticeably high or low pitched.

High-pitched bronchial breathing is typically heard over consolidated lung tissue in lobar pneumonia and is sometimes called *tubular*: low-pitched bronchial breathing may be heard over moderately large cavities and is hence sometimes called *cavernous*. These variations in pitch depend on whether the sound is conveyed to the ear through consolidated lung from small or large air passages. A special variety of bronchial breathing is known as *amphoric*. It resembles the sound produced by blowing across the mouth of a bottle, and consists of one or more low-pitched fundamental tones and a number of high-pitched overtones. It is characteristic of a direct communication between a bronchus and either a considerable cavity with smooth walls or a pneumothorax. The distinction from cavernous breathing is subtle and often academic.

When breath-sounds in a superficial bronchus can be heard through normal lung, the sound of the breathing combines both vesicular and bronchial elements. This variety of breath-sound is known as *broncho-vesicular*, and it is usually the expiratory sound which has more of a bronchial character. It may occur in health near the roots of the lungs behind; in the upper portions near the middle line in front; and especially

at the right apex for a few centimetres below the clavicle in front and above the level of the spine of the scapula near the mid-line behind. In the same areas, vocal fremitus and resonance may be increased, and some degree of whispering pectoriloquy may be heard. These findings, which sometimes lead to a mistaken diagnosis of disease at the right apex, are due to the fact that the trachea lies in immediate contact with the apex of the lung on the right side, whereas it is separated from it on the left by the aorta, the internal carotid artery, and the oesophagus.

The breath-sounds must be auscultated in the various regions that have already been examined by percussion, their character in each noted, and similar regions on the two sides of the chest compared, care being taken that the points examined correspond accurately to one another.

If the student understands how vesicular and bronchial breath-sounds are produced, he should have no difficulty in explaining the typical findings in disease. Vesicular breath-sounds may be present but reduced in intensity in any condition in which the entry of air to that part of the lung is diminished, as, for instance, in bronchopneumonia, where some alveoli are affected and others not. Breath-sounds of any kind may be diminished or absent where thickened pleura, pleural effusion, or pneumothorax interferes with or prevents the conduction of these sounds to the chest-wall. They may also be absent in any condition, such as collapse or fibrosis, in which no air enters or leaves alveoli, but at the same time the conditions necessary for the conduction of bronchial breath-sounds to the chest-wall are not fulfilled. Finally, bronchial breath-sounds of various kinds may be heard whenever patent bronchi are connected to the chest-wall by a sufficiently uniform sound-conducting medium. This occurs classically in the consolidation of lobar pneumonia, occasionally in the presence of a very large bronchial carcinoma, and in conditions previously mentioned.

### B. Vocal Resonance

The second series of observations is directed to *the intensity and character of the vocal resonance*. It varies in intensity even in health being more intense the nearer the stethoscope is to the larger bronchi. When the patient repeats the words 'one, one, one', or 'ninety-nine', the ear perceives, not the distinct syllables, but a resonant sound, the intensity of which depends on the loudness and depth of the patient's voice and on the conductivity of his lungs.

Each point examined on one side of the chest should be at once

compared with the corresponding point on the other side. Vocal resonance of normal intensity generally conveys the impression of being produced just at the chest-piece of the stethoscope. If it seems to be nearer the ear than this, the resonance is increased. When it appears to be near the ear-piece of the stethoscope the increase is marked, and the condition is often described as *bronchophony*.

If the words become clear and seem to be spoken right into the auscultator's ear, it will generally be found that whispered words are distinctly heard. This condition is called *whispering pectoriloquy*. Increased resonance occurs when the lung substance conducts the sound-waves set up by the voice more clearly than usual from the bronchi. Consolidation is the commonest cause. Bronchophony and whispering pectoriloquy occur when a moderately large bronchus is surrounded by a layer of solid lung reaching to the chest-wall, as in lobar pneumonia. Whispering pectoriloquy is also fairly characteristic of a cavity of some size communicating with a bronchus, and may be heard above the level of a pleural effusion. In some cases a certain degree of pectoriloquy is heard in health in the proximity of the trachea and large bronchi, and particularly at the right apex.

For reasons already explained, vocal resonance is either entirely abolished or much diminished where a layer of fluid separates the lung from the chest-wall (except when loud bronchial breathing is heard—see p. 158) and in pneumothorax. It is also diminished in cases of thickened pleura and of emphysema.

Above the level of a pleural effusion or in some cases over an area of consolidation, a nasal or bleating character may be imparted to the voice. It is known as *aegophony*.

### C. Added Sounds

These may arise in the lung or in the pleura. Sounds resembling pleural friction may be produced by movement of the stethoscope on the patient's skin, or of the observer's hands or clothes against the stethoscope. Sounds arising in the patient's muscles may resemble adventitious sounds and in particular the shivering of a cold patient makes any attempt at auscultation useless. The application of the stethoscope to hairy skin may produce sounds indistinguishable from crepitations. Sounds resembling coarse crepitations may also be heard over a broken rib.

The nomenclature of the added sounds arising in the lung has been confused since their original description by Laennec. He introduced the French word *râle* to describe any added sound heard in the chest.

When he wrote or spoke in front of patients, he used the Latin word *rhonchus* in the same connotation. As a result of confusion in translation, the terms are often used differently today. The least confusing classification of added sounds is to divide them into continuous *wheezing sounds*, or *rhonchi*, and interrupted *bubbling or crackling sounds*, or *crepitations*.

*Rhonchi*, which are prolonged uninterrupted noises, arise in the bronchi and are due to partial obstruction of their lumen, either by swelling of the mucosa or by viscid secretion. They may be high or low pitched, depending on whether they arise in small or large bronchial tubes. High-pitched rhonchi are called *sibilant* and have a squeaky quality, low-pitched rhonchi are called *sonorous*, and have a snoring quality. The former are most abundant during the later part of inspiration, and the latter may be heard during both inspiration and expiration and may be almost continuous. They may also be palpable. Rhonchi are characteristic of bronchitis and asthma, and in the latter condition are high-pitched and expiratory. When they are localized to one side of the chest, the possibility of a localized obstruction of a bronchial tube should be considered, as for example in bronchial carcinoma. The word 'broncho-spasm' as a description of a physical sign should be avoided.

*Crepitations* are discontinuous crackling or bubbling sounds which may be produced in either the alveoli, the bronchi, or the cavities. They sound like the bursting of air bubbles, and indicate the presence of fluid secretions. They may be classified as fine or coarse.

*Fine crepitations* are probably caused by the opening up of collapsed alveoli. The separation of the walls is accompanied by a clicking sound and when this condition occurs in a number of alveoli, the combined effect is to produce a sound of fine crepitation, which can be imitated by rolling a few hairs between the finger and thumb in front of the ear. It occurs only near the end of inspiration, and indicates the presence of exudation in the alveoli of the affected part of the lung. Fine crepitations are characteristically present during the first stage of pneumonia, at the apices in tuberculosis and at the bases in heart failure.

*Coarse crepitations* are bubbling or clicking noises which can probably arise in many sites, including large and small branches of the bronchial tree, or in cavities, and may be heard at almost any phase of, or during the whole of, respiration. They are often present in bronchitis in association with rhonchi: however, when they are restricted to the lung bases, the possibility of bronchiectasis or an interstitial pulmonary fibrosis should be considered.

A few crepitations may be heard in health, particularly at the lower borders of the lungs. These are abolished if the patient is asked to cough and are of no significance. In other cases crepitations are intensified after a cough or may only then make their appearance. Crepitations brought out by coughing are known as *post-tussive crepitations*. They are an important sign of tuberculous infiltration, and may also be heard over cavities.

The most common adventitious sound arising in the pleural cavity is a *friction-sound* or *pleural rub* characteristic of pleurisy at the stage when exudation is not abundant enough to separate the inflamed and roughened surfaces. It has a creaking or rubbing character, often quite characteristic, but sometimes rather hard to distinguish from crepitation. The friction-sound may be fine or coarse. In some instances it is palpable, but since coarse crepitations may be so too, this does not distinguish them. The chief features of difference are that friction-sounds occur during that part of inspiration when the roughened surfaces are rubbing against each other, to reappear at a corresponding period of expiration. They are, moreover, unchanged after the patient has coughed, whereas crepitations may alter under these conditions, because of changes in the disposition of the secretion which causes them. Friction is sometimes more localized than crepitations. Sometimes friction is intensified by pressing hard with the stethoscope. This causes the roughened surfaces to rub against each other more firmly. Pressure does not affect the intensity of crepitations. The situation of the doubtful sound, the presence of pain, or some point in the history of the case, may help in arriving at the diagnosis.

Finally there are certain manifestations of pulmonary disease which should be looked for outside the chest itself. These include clubbing of the fingers and cyanosis (both mentioned in previous chapters), and enlargement of cervical or axillary lymph nodes or the liver from secondary carcinoma. Respiratory failure with carbon dioxide retention (p. 170) may be associated with certain neurological signs. These include a coarse flapping tremor of the hands (*asterixis*), generalized twitching movements, and even convulsions. Such patients may become drowsy or stuporose, and this is aggravated by sedatives or injudicious oxygen therapy. In some cases there is papilloedema, which in a few instances may even be followed by optic atrophy.

## VI THE SPUTUM

Sputum may be mucoid, purulent, or frothy. Any of these

varieties may contain blood, or the sputum may consist entirely of blood.

*Mucoid* sputum occurs characteristically in chronic bronchitis when secondary infection is not present. It is clear, tough and sticky and usually scanty. Particularly tenacious sputum may be found in asthma. This may block the bronchi by forming plugs, or else be coughed up as 'casts' of the bronchial tree—during or after an attack; alternatively, sticky particles like sago—the 'perles' of Laennec—may be expectorated after an attack.

*Muco-purulent* sputum is seen in bronchitis (or other upper respiratory infections) when secondary bacterial infection has occurred: the sputum in bronchitis may also be frankly purulent.

*Purulent* sputum is thick and yellow (or green) and not sticky. It may occur in any condition in which infection is present, and is characteristic of bronchiectasis, broncho pneumonia and lung abscess. In bronchiectasis and lung abscess it may be copious and its expectoration readily influenced by change of posture.

*Frothy* sputum, characteristic of pulmonary oedema, may be white or pink and is often copious.

*Blood* may be coughed up alone, or the sputum may be more or less blood-stained. It must be distinguished from blood brought into the mouth from epistaxis or haematemesis. Its brighter colour and its frothy appearance usually makes its origin obvious. Further, patients who have had a haemoptysis commonly bring up blood-stained sputum for a day or two, while bleeding from the upper intestinal tract is characteristically followed by melaena. Haemoptysis may be due to pulmonary causes, including tuberculosis, bronchiectasis, pulmonary embolus, and carcinoma; to cardiac causes, including mitral stenosis; and very rarely to aneurysm of the aorta.

Several diseases cause a characteristic colouration of the sputum. In pneumonia it may be rusty and so viscid that it often will not fall out of an inverted spittoon; it is bright-yellow or green when a liver abscess has ruptured into the lung, and the latter colour also appears in some cases of pneumonia. When an amoebic hepatic abscess has discharged into the lung the sputum has the appearance of anchovy sauce.

The quantity of sputum coughed up in twenty-four hours is important; and especially whether change of position produces a large quantity or whether the sputum is produced in small amounts in any position.

The odour of the sputum is rarely important, but it may be putrid in bronchiectasis or lung abscess.

### Microscopical Examination of Sputum

The principal value of microscopical examination of the sputum is in the detection of bacteria and in the recognition of malignant cells. Eosinophils may be found in the sputum in allergic conditions, e.g. some cases of asthma; and in pneumonitis due to parasitic worms and in aspergillosis.

The methods used for the detection of bacteria, including the tubercle bacillus, are described on p. 354.

Malignant cells may be seen in the sputum, particularly in patients with squamous carcinomata of the main bronchi. Skill is required to differentiate them from epithelium and other cellular debris, but their appearance is sometimes unmistakable (Plate XIX).

Less common constituents of the sputum include fungi and yeasts, and golden-yellow asbestos bodies in asbestosis (Plate XIX).

## VII X-RAY EXAMINATION

Radiological examination of the chest is most important, because many localized and even some widespread infiltrative lesions (e.g. sarcoidosis) may produce no abnormal physical signs. In the early diagnosis of tuberculosis and carcinoma, it is paramount. Serial X-rays form an integral part of the estimation of progress in many chest diseases. A brief outline of the standard methods will be given here.

1. Radiography (postero-anterior and lateral films).
2. Screening.
3. Bronchography.
4. Tomography.

### Radiography

The ordinary standard X-ray film of the chest is a *postero-anterior* view, that is to say one taken with the film against the front of the patient's chest and the X-ray tube two metres behind the patient. It is examined systematically on a viewing box in every case. The following is a simple plan of examination.

(i) **The bony skeleton.** Is the chest symmetrical? Is any scoliosis present? Are the ribs unduly crowded or widely spaced in any area? Are cervical ribs present? Are the ribs eroded or do they appear the site of malignant deposits?

(ii) **The position of the patient.** Is the patient straight or rotated? If straight, the inner ends of the clavicles will be disposed symmetrically with reference to the vertebral column.

(iii) **The position of the trachea.** This is seen as a dark column representing the air within the trachea. The cartilagenous rings are not visible. Is it centrally placed or deviated to one or other side?

(iv) **The outline of the heart and mediastinum.** Is this normal in size, shape, and position?

(v) **The diaphragm.** Can the outline of the diaphragm be seen on each side, and is it normal in shape and position? Are the cardio-phrenic and costophrenic angles clearly seen?

(vi) **The lung fields.** For radiological purposes these are divided into three zones:

*Zone i (Upper Zone)* extends from the apex to a line drawn through the lower borders of the anterior ends of the 2nd costal cartilages.

*Zone ii (Mid Zone)* extends from this line to one drawn through the lower borders of the 4th costal cartilages, and contains the hila of the lungs.

*Zone iii (Lower Zone)* extends from this line to the bases of the lungs.

Each zone is systematically examined on the two sides, and any area which appears abnormal is carefully compared with the corresponding area on the opposite side. The minor interlobar fissure, which separates the right upper and middle lobes, may sometimes be seen running horizontally in the 3rd and 4th interspace on the right side. The major interlobar fissure, which separates the lower lobes from the remainder of the lungs, is not seen in a normal postero-anterior film.

*Lateral views* are indispensable in the localization of lung lesions, for the postero-anterior view does not show whether a shadow is situated in the anterior or posterior part of the chest, or (if in the mid-zone) whether in the upper or lower lobe.

The following is a simple plan of examination:

(i) **The bony skeleton.**

(ii) **The position of the trachea.**

(iii) **The diaphragm.** As the level differs on the two sides, a double outline may be seen, that of the side nearer the film being the clearer.

(iv) **The lung fields.** These are obscured by two relatively opaque

areas, one above and behind, due to the shoulder joint, and one below and in front, due to the heart, which rests on the anterior part of the diaphragm. There are thus left two relatively clear areas—one above and in front, behind the upper part of the sternum, and one below and behind, including the angle between the diaphragm and the spine.

In the lateral views, the interlobar fissures are more often seen. Their normal positions have already been described (p. 146). Their recognition is useful both in localizing lesions and in detecting shrinkage of a lobe from fibrosis or collapse.

### Screening

This procedure is used mainly to detect abnormalities of the heart and paralysis of the diaphragm. By standing the patient in different positions, it is possible to see enlargement of the various chambers of the heart and main vessels and paradoxical pulsation of the left atrium. Enlargement of the left atrium is detected by noting displacement of the barium-filled oesophagus. When diaphragmatic paralysis is present *paradoxical* movement is seen when the patient coughs or sniffs, that is, the diaphragm ascends when it should descend, and vice versa.

### Bronchography

For this purpose a radiopaque iodized oil (usually Dionosil Oily) is introduced into the trachea and allowed to run into the bronchi. X-ray pictures are taken and the corresponding bronchi are clearly outlined. With suitable manipulations, the whole of the bronchial tree can be outlined, but more than one sitting may be required.

The oil may be introduced:

- (i) by passing a special needle through the crico-thyroid membrane, after suitable anesthetization of the needle-track and the mucous membrane of the trachea (this method is the one most usually adopted nowadays);
- (ii) by means of a catheter passed through the nasal cavity into the trachea;
- (iii) directly into the larynx over the back of the tongue;
- (iv) through a bronchoscope (this method is used only occasionally, and for special purposes).

If the bronchi on both sides are to be outlined at one sitting, the side thought to be abnormal should be filled first, and postero-anterior and lateral views taken. The opposite side can then be filled

and a further postero-anterior view taken. The patient should be warned not to attempt to swallow food or drink till the effect of any local anaesthetic given has worn off.

### Tomography

An ordinary X-ray picture consists of shadows at all depths in the chest, superimposed on one another. It has the disadvantage that not more than some 40 per cent of the lung tissue is shown without its being obscured by shadows of the bony thorax or of mediastinal contents. The tomograph is a device whereby a picture is obtained of a section of the thorax at any given depth. The tube and plate are moved in the arc of a circle as the exposure is made, in such a manner that the structures in one section only remain in focus and anything out of the plane of this section is blurred out. Sections can be taken at different depths in the chest, as desired, and so the appearances in chest X-rays can often be greatly simplified. It is mainly used to detect cavitation in apparently opaque shadows in the lung fields, and to give a clearer picture of hilar shadows.

## VIII BRONCHOSCOPY AND THORACOSCOPY

By means of the bronchoscope, the main bronchi and their branches can be directly inspected, small portions of tissue can be removed for biopsy, and therapeutic procedures carried out. The main value of bronchoscopy is in the diagnosis of carcinoma of the bronchus and in deciding whether this is operable. After an artificial pneumothorax has been induced, the pleura can be inspected with the aid of a thoracoscope, and further assistance in diagnosis may be obtained. These are specialized surgical techniques.

## IX PLEURAL ASPIRATION AND BIOPSY

**Pleural effusions** may be drained by inserting a wide-bore needle into the fluid-filled pleural space through one of the rib spaces. This is done under local anaesthesia, and the needle is inserted perpendicular to the skin and pleura, just above a rib margin. Aspirations may be *therapeutic*, to relieve respiratory embarrassment due to a large effusion, or *diagnostic*, to remove fluid for examination.

**Pleural fluid** can be examined macroscopically and microscopically. Full details cannot be given here, but the colour, consistency, and

quantity should be noted. A *transudate* from the capillaries, as occurs in cardiac and renal disease, can be distinguished from an *exudate* resulting from pleural inflammation by its lower protein content ( $> 3$  g. per 100 ml.) and specific gravity (usually  $< 1.015$ ). Frankly blood-stained effusions occur with carcinoma, pulmonary infarction, or trauma. In tuberculous effusions, the fluid is straw-coloured and copious (often well over 1 litre), and may coagulate on standing; under the microscope many leucocytes are seen, lymphocytes often predominating. Tubercle bacilli are rarely seen, but can more commonly be cultured. In other inflammatory exudates, many polymorphs are seen. In *empyema* pus is aspirated which may be full of white cells and organisms.

Needle biopsy of the pleura may also be useful in establishing the diagnosis of the cause of a pleural effusion. In a high proportion of patients the presence of carcinoma or tuberculosis may be detected.

## X LUNG FUNCTION TESTS

In recent years, tests of lung-function of increasing complexity have been introduced, which are beyond the scope of this chapter; but some of the simple tests useful in clinical practice will be described. It must be stressed that lung-function tests enable the clinician to make a *physiological* rather than a *pathological* diagnosis. That is to say, they will, for instance, tell one that there is obstruction to air-flow, but not that the patient has a bronchial carcinoma. Also, they are more likely to be abnormal if there is a diffuse process affecting the lung than if there is merely a localized lesion. They may be useful under the following circumstances:

1. To give an objective assessment of a patient's disability.
2. To follow the progress of a disease and the effect of treatment.
3. To try to differentiate possible causes of a patient's dyspnoea.
4. To aid the management of cases of respiratory failure.

**1. Vital capacity and spirometry.** The simplest and still perhaps the most valuable tests are the measurement of the patient's *vital capacity* and the recording of the *expiratory spirogram*. These can be measured by various types of *spirometer* and recorded graphically (Fig. 45). The patient inhales maximally and then breathes out as hard and as fast as he can. Measurements are then made of the amount of air he expels and the speed at which he does so. The amount of air expelled by maximal voluntary effort is the *vital capacity*. In normal subjects

three quarters of this air is expelled within the first second and all within about 3 seconds. Thus the normal curve is a steep one. In diseases such as asthma, bronchitis, and emphysema there is obstruction to the flow of air out of the lungs, owing to collapse or constriction of the airways, or intraluminal obstruction, owing to mucus or

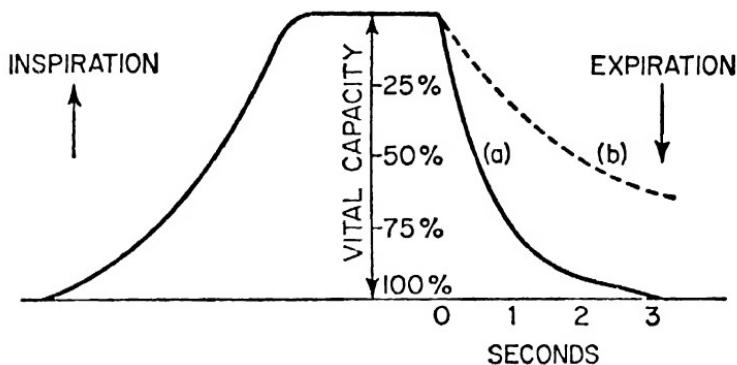


Fig. 45. The expiratory spirogram. A, normal; B, obstructive airways disease.

oedema, so that the curve is flattened (Fig. 45). On scrutiny of the curves, this flattening is apparent, but a numerical value can be given to the degree of flattening by recording the volume expired in one second. This volume (Forced Expiratory Volume at one second, or FEV<sub>1</sub>) is then expressed as a ratio of the *Vital Capacity* (VC). This ratio

$$\frac{\text{FEV}_1}{\text{VC}}$$

is called the FEV percentage and should exceed 70 per cent in healthy individuals under 60 years of age. If the percentage is below this figure, the patient may be described as suffering from *obstructive-airways disease*. The test may be repeated after the use of a bronchodilator aerosol, and if the FEV<sub>1</sub> improves, the *airways obstruction* is said to be *reversible*. Broadly speaking, in asthma and bronchitis the obstruction is partly reversible by bronchodilators, whereas in emphysema this is not so. However, the distinction between these conditions is not as clear as might be supposed. The *Residual Volume* (RV), or amount of air still trapped in the lungs after maximal expiration, can be measured by a more complicated test, and so the *Total Lung Capacity*

computed. Both the *Residual Volume (RV)* and its ratio to the *Total Lung Capacity* are increased in obstructive-airways disease.

**2. Diffusion studies.** Having entered the lungs, air is normally fairly evenly distributed throughout them. This distribution may be quite uneven in disease, but complex apparatus is needed to detect this.

Oxygen and carbon-dioxide must pass freely between alveoli and pulmonary capillaries. The passage of these gases occurs largely by diffusion. Carbon-dioxide diffuses very readily, and rarely presents a problem; but in certain conditions, notably the *interstitial fibroses*, and some pulmonary infiltrations, such as *sarcoidosis*, the diffusion of oxygen may be limited. Unequal distribution of inspired gas in relation to *perfusion* of the pulmonary capillaries may also cause abnormal oxygenation of the blood. The *Diffusing Capacity* or *Transfer Factor* of the lungs is measured by using *carbon-monoxide* as a marker gas, and the uptake of this gas is measured either during a single period of breath holding or by a steady-state technique. A reduced *Diffusing Capacity* or '*diffusion block*' can cause reduced oxygenation of the arterial blood, particularly on exercise.

**3. The blood gases.** The overall function of the lungs is to ventilate the blood adequately— that is to say, to maintain limits needed for normal tissue-metabolism. In *respiratory failure* these physiological limits are exceeded. In particular, carbon-dioxide is inadequately eliminated from the body, and so its level in the blood rises in the same way as the blood-urea rises in renal failure. If the  $\text{PCO}_2$  is elevated above 45 mm. Hg. we can say that the patient is in respiratory failure due to *alveolar hypventilation*. If the  $\text{PCO}_2$  rises further, clinical features of carbon-dioxide retention appear, and urgent treatment is required. In some instances, repeated estimation of  $\text{PCO}_2$  may provide a valuable guide as to the efficacy of treatment which is given.

Direct measurement of the  $\text{PCO}_2$  in arterial blood cannot always be made. Fortunately an alternative method, requiring neither arterial blood nor complex electrodes, has been introduced by Campbell and Howell. In principle this involves the patient rebreathing into a bag until the gas-mixture in the bag is in equilibrium with alveolar air and hence indirectly with arterial blood. The final gas-mixture in the bag is then analysed for carbon-dioxide, using a simple apparatus.

The nomenclature of these measurements is complex, and reference should be made elsewhere for a fuller explanation. Normally the

*arterial oxygen saturation* is above 97 per cent, the *carbon-dioxide tension* ( $\text{PCO}_2$ ) is 37–43 mm. Hg, and the pH 7.38–7.42. The concept of  $\text{PCO}_2$  is an important one, as it is the *tension* or *partial pressure* of a gas that determines its diffusion between the tissue and capillary blood. Oxygen *saturation* is still used, largely because methods of measuring oxygen tension in blood have only very recently been developed and are still difficult.

**4. Other tests.** Two other simple measurements that may be useful are the *Minute Volume* and the *Maximum Voluntary Ventilation*. The *Minute Volume* may be calculated by multiplying the *Tidal Volume* (the volume of each breath) by the *Respiratory Rate*. It is usually measured by means of a spirometer or by collecting expired air into a bag. In either case breathing through a mouthpiece into a system presenting some resistance to air-flow may cause the subject to increase his respiration. This effect may be diminished by using a low-resistance portable Wright's respirometer. The *Minute Volume* only measures air entering or leaving the respiratory tract at the mouth. Because the conducting airways (trachea, bronchi and bronchioles) act as a respiratory 'dead space' where no gas-exchange occurs, the *Alveolar Ventilation* is always less than the *Minute Volume* (though proportional to it). Direct measurements of 'dead space' are not often made, and the adequacy of alveolar ventilation can be determined by estimation of the  $\text{PCO}_2$ .

In the *Maximum Voluntary Ventilation Test* the subject breathes as hard and as fast as he can into a spirometer for a 15-second period. The volume of air he breathes during this period is multiplied by four, to convert it to litres per minute. This test is dependent on several factors: (1) the full co-operation of the patient; (2) the neuromuscular function of the muscles of respiration; and (3) the presence or absence of airways-obstruction. It is therefore not a test of any *single* aspect of lung function. It may serve as a guide as to what the patient is capable of achieving by voluntary effort.

## 7 THE URINE

### Collection of Samples

For most purposes a specimen of urine as passed into a clean glass vessel is sufficient. Because of the danger of introducing infection, catheters should be avoided, unless absolutely necessary. If the urine is required for bacteriological purposes, a mid-stream specimen should be collected with sterile precautions after the vulva or glans penis has been cleaned with an antiseptic solution, such as 1 per cent aqueous centrimide.

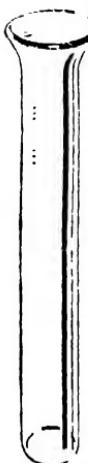
The following method of obtaining midstream specimens from females has been found to give bacteriological results comparable with those obtained by catheterization. It employs glass tubes with a bell-shaped end which are supplied by Philip Harris Ltd, 144 Edmund Street, Birmingham 3 (Fig. 46).

The subject sits on a low stool, or, if in bed, on a low bed-stool, with the legs apart. The labia are separated, and the vulva is cleansed with an aqueous 1 per cent solution of cetrimide and dried with sterile gauze.

A sterile tube of appropriate size is chosen and the bell end is placed in position over the urethral orifice. The tube is held gently in position by the nurse, and the labia allowed to fall back over the tube. The patient then voids urine and the mid-portion of the specimen is collected in a sterile jar.

This method is simple and can easily be carried out by one operator; contamination of the specimen by contact with the labia is avoided; and it is possible to obtain specimens while menstruation is in progress.

Any suspended matters soon settle to the bottom of the glass, and the examination of the sample may then be conducted (1) physically; (2) chemically; (3) microscopically.



**Fig. 46. Glass tube for taking sterile specimens of urine from female patients. These tubes are supplied in 10 cm. and 15 cm. lengths, approximately 1.5 cm. diameter.**

## I PHYSICAL EXAMINATION

Attention should be paid to the following points: (1) quantity; (2) colour and transparency; (3) specific gravity; (4) naked-eye characters of the deposit.

### 1 Quantity

The normal quantity of urine passed daily varies widely—from 700 to 2500 ml.

Normally, very much more urine is secreted during the day than during the night. Approximation of the night quantity to that of the day is always abnormal, and occurs especially in renal failure, of which it may constitute one of the earliest signs. Thus, nocturnal polyuria—a symptom which is now often referred to by the unfortunate term '*nocturia*'—may be the first symptom of failure of the concentrating power of the kidney and may indicate commencing renal failure.

An *increased secretion* of urine occurs physiologically after increased consumption of food or drink, and after exposure to cold. Conversely, *diminished secretion* occurs when little food or drink has been taken, and after exposure to heat—especially if followed by sweating.

A *pathological increase* is an early sign of chronic renal failure. Polyuria is also a feature of both types of diabetes, and accompanies the elimination of oedema fluid. *Abnormal diminution* of urine follows sudden lowering of arterial pressure, as in shock, but is more commonly due to reduction of glomerular blood flow by inflammation, as in acute nephritis; or by dehydration, as in fever, diarrhoea, and vomiting; or in terminal heart failure. With severe disturbance of renal circulation, anuria may result.

### 2 Colour and transparency

Normal urine has the colour of amber or pale sherry. The exact tint fluctuates widely.

Small quantities of blood give the urine a smoky appearance and larger quantities make it brownish or red. Haemoglobin in large quantities, as in blackwater fever, give it a dark-red (port-wine) colour, to one that is brownish-black or almost black. The presence of bile gives it a brown or even dark brown colour. The urine is abnormally pale when it is very dilute, i.e. with specific gravity

## THE URINE

of 1002 or 1003, and in renal failure, when the normal colouring matter (urochrome) is greatly diminished or absent.

## CHIEF VARIETIES OF ALTERATION IN COLOUR OF URINE, WITH THEIR CAUSES

<i>Colour</i>	<i>Cause</i>	<i>Condition or remarks</i>
NEARLY COLOURLESS	Large amount of urine excreted	Much drinking. Diabetes mellitus or insipidus
DARK AMBER	Small amount of concentrated urine	Hard muscular work Fever
ORANGE-COLOURED	Excess urobilin	Haemolytic anaemias
ORANGE-COLOURED	Administration of rhubarb, senna, cascara	(These turn yellow with acid, red with alkali; a normal colour going red with alkali=phenolphthalein)
RIDDISH-BROWN		
PINK	Phenylindandione (Dindevan)	
BROWN	Bile Furazolidone (Furoxone) Niridazole (Aambilhar)	Jaundice 'Coca-Cola wine'
RED	i Blood (p. 179) ii Haemoglobinuria iii Aniline dyes in sweets iv Excessive consumption of beetroot. Anthocyanuria	
PORT-WINE BROWNISH-BLACK	Porphyria i Melanin ii Methaemoglobin iii Much haemoglobin iv Alkaptonuria	(p. 180) Melanotic sarcoma (Yields chocolate deposit) Urine colourless when passed and darkens on standing
GREENISH-BLACK	Phenol and lysol poisoning	May reduce Fehling's solution. Ferric chloride test may be positive
YELLOWISH-GREEN	Santonin	(Turns red with alkali)
GREEN		
GREENISH-BLUE	Administration of methylene blue	(Violet with alkali; cuts off red and yellow in spectrum)
YELLOWISH AND MILKY	i Pus ii Fat	(p. 187) Chyluria

Normally, when freshly passed, urine is quite transparent, but it may be *opalescent* from the presence of various substances in suspension, of which the most important are pus, bacteria, and phosphates. Phosphates disappear on adding acid. If the opalescence persists after filtration, it is due to the presence of bacteria. Urine that has cooled may be cloudy from the presence of urates. In this case it will become clear again when warmed.

### 3 Specific Gravity

The specific gravity of urine is measured by a *urinometer*. An ordinary urinometer is graduated for a temperature of 15°C, and will record variations in a specific gravity from 1000 up to 1060. The calibration of the urinometer should be checked by seeing that it reads 1000 when placed in pure water. Care must be also taken to read the true level of the surface of the urine and not the edge of the rim which heaps up around the shaft of the urinometer. The normal specific gravity varies from 1001 to 1025, depending on the state of hydration and the time of day. It may occasionally rise to 1035 even in health.

The specific gravity is greatly increased by cooling. If, for example, it is 1020 when passed, it will rise to about 1025 when cooled to the temperature of the room. This examination should, therefore, be made when the urine has cooled to room temperature.

In normal urine the specific gravity is in direct proportion to the amount of urea and chlorides present. An abundant urine of high specific gravity is characteristic of diabetes mellitus. In diabetes insipidus, on the other hand, the specific gravity may fall to nearly that of distilled water, and this may also happen in hysterical polydipsia. The presence of protein in the urine also affects its specific gravity, 1 per cent increasing the specific gravity by three points.

Serial examinations of the specific gravity constitute the simplest and one of the most valuable methods of observing renal function. With normal kidneys the concentration of the urine varies to a considerable extent. As renal failure develops the specific gravity approximates more closely to 1010 (or 1006 if the urine is tested while still warm). With complete loss of concentrating power the specific gravity becomes fixed at 1010, the urine then being isotonic with the plasma. The specimen passed on rising should always be tested for this purpose, as it is normally the most concentrated that

is passed in the day. In a normal person the specific gravity should be at least 1020, if no fluid has been drunk for nine hours.

#### 4 Naked-eye Characters of the Deposit

When voided, normal urine is perfectly clear and transparent. After it has stood for some time there appears in it a deposit of 'mucus'. This forms a woolly-looking cloud, which usually settles to the bottom of the glass, but if the urine is of high specific gravity, it may be in the middle of the glass, or even at the top. If traces of blood are present in the urine, the cloud of 'mucus' has often a brownish tint.

The normal urinary ingredients, which may separate out in the form of a deposit visible to the naked eye, are phosphates, urates, and uric acid.

Phosphates separate out as a white deposit when the urine is neutral or alkaline and especially when it is heated. The deposit is dissolved by acetic acid. Urates and uric acid form yellow, red, or brown deposits, particularly if the urine is concentrated or highly acid. Although usually of no significance, they may appear after irradiation of the spleen in leukaemia or related diseases and may cause renal colic or even urinary obstruction.

### II CHEMICAL EXAMINATION OF THE URINE

#### 1 Reaction

It is customary routinely to test the reaction of the urine with litmus paper, but the result is rarely important except when a drug has been given with the intention of altering the pH for therapeutic purposes. Normal urine is nearly always acid. Rarely it is repeatedly neutral or alkaline, when the patient is not taking alkalis, and this may indicate impairment of the power of the tubules to excrete acid. This can be confirmed by accurate measurements of the pH of the urine after the patient has taken ammonium chloride.

#### 2 Examination of the Urine for Chlorides

Sodium chloride is the chief inorganic constituent of normal urine. Small quantities of the potassium salt also occur. The following is a simple test for measuring the approximate concentration of sodium chloride in the urine.

The solutions required are:

20 per cent potassium chromate solution.

2·9 per cent silver nitrate solution.

Ten drops of urine are measured with a pipette into a small test-tube. The pipette is rinsed and one drop of the potassium chromate solution is added. The pipette is then rinsed again and the silver nitrate solution is added drop by drop, the test-tube being shaken after each addition, until the colour changes suddenly from yellow to brown. The number of drops of silver nitrate used to reach the end-point gives the concentration of chlorides in the urine expressed as grams of sodium chloride per litre of urine. A control test with distilled water should be performed to make sure that the chromate solution is not contaminated with chloride.

The amount of sodium chloride per litre depends on the concentration of the urine. The average amount excreted is about 5 to 15 g. in the 24 hours. With urine of specific gravity of 1020 or more, less than 3 g. of sodium chloride per litre suggests salt depletion. This test is useful in the tropics, when salt depletion from extreme sweating is common, particularly in the diagnosis of heat exhaustion from other causes of collapse. In temperate climates it can be employed in medical and surgical cases complicated by continued excessive sweating, diarrhoea, or vomiting to detect salt deficiency; and during intravenous saline administration, to indicate when a deficiency of salt has been restored, but blood-electrolyte estimations are to be preferred when available, because the kidneys may under certain circumstances lose their ability to excrete salt, so that the urinary findings may be misleading.

### 3 Abnormal Chemical Constituents of Urine

#### Proteins

Before proceeding to apply *tests for protein* it is essential that the urine should be absolutely clear. It may therefore be necessary to filter it. If, after filtering more than once, the urine remains turbid, bacteria are probably present, and can be removed by long centrifugation, or by shaking up the urine with powdered barium carbonate and filtering. If the turbidity is due to urates, it will disappear when the urine is heated. The following tests should then be proceeded with.

**1. Sulpho-salicylic acid test.** This test for protein is very reliable and does not require heat. In a test-tube place about 5 ml. of urine.

Filter if cloudy, add 6 drops of 20 per cent sulpho-salicylic acid. The formation of a cloud indicates the presence of protein. The cloud is seen best when looked for against a black background.

**2. Boiling test.** This is a satisfactory test for protein, but needs to be carried out with care.

Fill a small test-tube two-thirds full of urine. If the urine is alkaline, add a small piece of litmus paper to the urine and add 10 per cent acetic acid drop by drop, mixing thoroughly after each drop until the litmus paper is just red. Incline at an angle, boil the top inch over a flame holding the bottom of the tube, and examine against a dark background. A cloudiness indicates the presence of either protein or phosphates. Add 10 per cent acetic acid drop by drop, and boil. If the cloud disappears, it consists of phosphates; if it persists, protein is present. Acid should be added drop by drop till no further precipitation of protein occurs. If there is more than a light cloud, add a few more drops of 10 per cent acetic acid, mix well, and, holding the test-tube in a holder, boil the whole volume. The test-tube is put aside for  $\frac{1}{2}$  hour or more for the protein to settle. This test is not very reliable if the urine is of extremely low specific gravity.

**Quantitative estimation of protein.** This can be done with sufficient accuracy for clinical purposes by means of Esbach's albuminometer. The principle of the method consists in measuring the depth of the coagulum produced in the urine by the addition of picric acid. The instrument consists of a thick glass test-tube, with graduations on it from 0 up to 7. To make up Esbach's reagent, dissolve 10 g. of picric acid and 20 g. of citric acid in about 900 ml. of boiling water. Allow this to cool and add water to 1 litre.

**Method.** Filter the urine if not already clear, and if alkaline render slightly acid with acetic acid. If the specific gravity be 1010 or more, dilute the urine sufficiently to bring the density below that level (to 1008). This is important, and is often overlooked. Fill the tube with the urine up to the mark U. Pour in the reagent up to the mark R. Close the tube with a rubber stopper, and gently invert it a few times to allow the fluids to mix. Set aside for twenty-four hours. At the end of that time read off the level of the surface precipitate. The figures on the scale represent grams of dried protein per litre of urine.

Divide by 10 to get the percentage. If the urine requires to be diluted, the result must, of course, be multiplied the requisite number of times.

This method yields only approximate results, since the precipitates obtained in different urines vary in compactness and in the length of time they take to settle. One should also remember that it measures only the concentration of protein in the urine and not the total amount.

Very small quantities of protein cannot be estimated by Esbach's method,

as the instrument does not record less than 0·1 per cent. If after the first trial the level of the precipitate is found to be above the mark 4, the urine must be diluted and a fresh estimation made.

For ordinary purposes it is often sufficient to express the amount of protein present as a faint trace, a trace, a cloud, a heavy cloud, or, if there is enough to form a precipitate, to express the height of the precipitate as a fraction of the height of the whole column of urine, when the whole tube has been boiled, and the precipitate allowed to settle. If the fraction is one half, the urine contains about 1 per cent of protein, and if one eighth, about 0·2 per cent. A 'cloud' of protein represents only about 0·02 per cent.

Proteinuria may be found in disease of the kidneys or any other part of the renal tract. For its significance textbooks of medicine must be consulted, but a few relevant points may be mentioned here. Slight proteinuria in the female can be regarded as significant only when precautions have been taken to avoid contamination by vaginal secretions. If kidney disease is suspected, but protein is absent on repeated chemical examinations, and blood, pus, or casts are not found on repeated microscopical examinations, there is no renal damage and elaborate renal efficiency tests are superfluous. If protein is present in the urine, in the absence of other signs of disease in the urinary tract, it may be due to 'benign', 'postural', or 'orthostatic' proteinuria. In this condition protein is secreted in the upright but not in the horizontal position. The patient should therefore empty the bladder immediately before he gets into bed, and a specimen should be passed as soon as he rises in the morning. If there is no protein in this specimen, the presence of protein in other specimens passed during the day is of no pathological significance.

**Bence Jones proteose** is usually, but not invariably, found in the urine of patients with multiple myelomatosis a fatal disease involving the bone-marrow. This proteose is also found, very rarely, in the urine of patients with leukaemia. It coagulates at a lower temperature (55°C and under) than the usual coagulable proteins of urine. Typical Bence Jones proteose has also the remarkable property of going into solution again at about 100°C, if, as is usually the case, the concentration of salts in the urine is suitable.

### Blood and its Derivates in Urine

Blood may appear in the urine as a whole (haematuria), or blood-pigment may appear without corpuscles (haemoglobinuria). These two conditions can only be differentiated by examining the deposits for blood cells. The detection of small numbers of red cells, such

as may be found for several days after an attack of renal colic, can only be achieved by microscopic examination (see p. 187).

If urine contains only a small amount of blood or blood-pigment it has a peculiar opaque appearance, to which the term 'smoky' is applied. Large quantities of blood give to the urine a red colour varying in intensity with the amount of blood present. The blood-corpuscles are apt to settle at the bottom, producing a flocculent deposit, which is brown or red according to the amount of the blood and the degree of its alteration.

The presence of blood or haemoglobin may be confirmed by the finding of red cells under the microscope: by the guaiac test, or more conveniently by the use of the Occultest tablets.

i. *Guaiac test.* Take 1 in. of urine in a test-tube, add to it two drops of tincture of guaiacum. A white precipitate forms, owing to partial precipitation of guaiacum resin. Now add 1 in. of ozonic ether without shaking. If blood pigment is present, a blue colour appears at the line of junction of the fluids.

ii. One drop of urine is placed in the centre of the test-paper provided and one *Occultest* tablet is placed on the resulting moist area. Two drops of water are run on to the tablet. If no blue colour appears in two minutes, the test is negative. The presence of blood in the urine is indicated by the appearance of a diffuse blue colour on the paper around the tablet within two minutes of the addition of the water.

**Porphyria.** Porphyrins occur normally in the urine in very small amounts, and may be considerably increased without affecting its colour. In the disease called porphyria, which follows the ingestion of sulphonal in some patients and is due to an inborn error of metabolism in others, they appear in the urine in large quantities and give it a dark port-wine colour. The guaiac reaction is negative and the presence of the porphyrins may be confirmed by spectroscopy, if they are present in any quantity. Such urine also shows fluorescence under Wood's light.

Urine which contains blood or haemoglobin also, of course, contains some protein, and it is often difficult to say whether the blood is sufficient to account for all the albumin present, or whether true albuminuria exists in addition. If human blood is added to normal urine in an amount sufficient to produce distinct smokiness, the quantity of protein amounts to merely a trace. Even when the quantity added is sufficient to render the urine distinctly red, the amount of protein is only  $\frac{1}{2}$  per 1000.

### Sugars in the Urine

Glucose is by far the most important of the sugars which may appear in the urine. In normal people it occurs in amounts too small to be detected by the usual methods employed, and if it is so detected its presence may be regarded as pathological. Glycosuria may be due to excessive blood glucose levels as in diabetes mellitus or to defective renal tubular re-absorption—'renal' glycosuria. The presence of a reducing substance in the urine may be detected by means of Benedict's test or by the use of Clinitest tablets.

*i. Benedict's test.* To 5 ml. of Benedict's reagent add 8 drops of the urine, boil for two minutes, and allow to cool. If a reducing substance is present, a precipitate will appear, varying from a light green turbidity to a red precipitate. If the reduction is due to glucose, the test gives approximately quantitative results:

A light-green turbidity	= 0·1 to 0·5 per cent of sugar
A green precipitate	= 0·5 to 1·0     "     "     "
A yellow precipitate	= 1·0 to 2·0     "     "     "
A red precipitate	= 2·0 per cent sugar or over

*ii. Clinitest.* This is a convenient modification of Benedict's test, in which the ingredients are present in a tablet and the necessary heat is provided by the interaction of sodium hydroxide and citric acid. Five drops of urine are placed in a test-tube with the dropper provided. The dropper is rinsed and ten drops of water are added. One Clinitest tablet is dropped into the tube and the resulting reaction observed. Effervescence occurs, followed by boiling. Fifteen seconds after the boiling has ceased, the tube should be shaken gently and the colour of the contents compared with the colour scale provided. When a reducing substance is present, the copper sulphate in the solution is converted to cuprous oxide, causing the colour to change through green ( $\frac{1}{2}$  per cent) to orange (2 per cent).

The reducing substances which may be found in urine are glucose, lactose, fructose, pentose, and homogentisic acid. Of these much the commonest and the most important is glucose. This can be detected by the use of *Clinistix* reagent slips. This is an enzyme test, the slip being impregnated with glucose oxidase, peroxidase, and orthotolidine. In the presence of glucose oxidase, glucose is converted into gluconic acid and hydrogen peroxide. The latter in the presence of peroxidase converts orthotolidine to its blue oxidized

form. The tip of the slip is dipped into the urine to be tested. If glucose is present, the moistened end turns blue, usually within one minute. The test is sensitive, and specific for glucose, but does not give a reliable indication of the amount present.

Homogenistic acid is present in the urine in alkapttonuria. Lactosuria occurs in late pregnancy and during lactation. Pentosuria is usually due to a rare inborn error of metabolism but may follow the ingestion of certain fruits. Its presence may be confirmed by Bial's reagent (1 g. of orcin, 500 ml. of HC1, specific gravity 1151, 25 drops of 10 per cent solution of ferric chloride). 5 ml. of this reagent are boiled in a test-tube and then removed from the flame, and 5 drops of urine are added. A green ring at the junction is diagnostic of pentose. It is most important to remove the tube from the flame, as otherwise other substances may give a positive result.

In practice, if a reducing substance is present in the urine and the patient has indubitable symptoms of diabetes mellitus, no further investigation is necessary. If no symptoms are present, it is often simplest to resort immediately to the sugar tolerance test (p. 373). If the blood-sugar curve is normal, the reducing substance in the urine is of no pathological significance.

### Bile in the Urine

Urine which contains bile is brownish-yellow to dark-brown in colour. The simplest test for the presence of bile in the urine consists in shaking it in a test tube and observing the colour of the froth. If this is yellow, bile-pigments are present in the urine. Of other tests which have been used for the detection of bilirubin in the urine, Fouchet's test is probably the best for the laboratory and Ictotest for the practitioner.

i. *Fouchet's Test.* 5 ml. of a 10 per cent solution of barium chloride are added to 10 ml. of urine, and the resulting mixture is filtered. The deposit on the filter paper is spread on another dry filter paper. One drop of Fouchet's reagent (trichloracetic acid 25 g., 10 per cent ferric chloride 10 ml., distilled water to 100 ml.) is added, and a blue or green ring indicates the presence of bilirubin.

ii. *Ictotest.* Five drops of urine are placed on a square of the test-mat provided. One Ictotest tablet is placed in the centre of the moistened area. Two drops of water are placed on the tablet. If bilirubin is present, the mat around the tablet turns bluish-purple. A pink or red colour should be ignored.

### Urobilinogen and Urobilin in Urine

Bilirubin secreted by the liver into the bile is reduced by the bacteria of the intestine to urobilinogen and urobilin. Some of this is reabsorbed and circulates in the blood-stream. Of this a small amount is excreted in the urine, but the majority under normal circumstances is re-excreted by the liver into the bile. Estimation of the urobilinogen and urobilin in the urine may therefore be useful in several circumstances. In obstructive jaundice, a complete absence of urobilinogen and urobilin indicates that the obstruction is complete and that no bile-pigment is reaching the intestine. In patients without jaundice an excess of urobilinogen and urobilin in the urine is due to the inability of the liver to excrete these substances into the bile and indicates hepatic dysfunction. Such an excess is often present in the pre-icteric stage of infective hepatitis and in diffuse diseases of the liver, such as severe cirrhosis. But the most important cause of an increase of urobilin and urobilinogen in the urine is excessive haemolysis.

*Method.* To 10 ml. of fresh urine add 1 ml. of Ehrlich's aldehyde reagent (para-dimethylaminobenzaldehyde 2 g. dissolved in hydrochloric acid 5 per cent 100 ml. Pure analytical hydrochloric acid must be used). After 3 to 5 minutes, normal urine shows a faint but distinct reddish tinge, much intensified by heating. If no red colour develops even on heating, urobilinogen is absent from the urine; if there is a distinct red colour in cold urine, it is present in excess. Normal urine shows no reddish tinge if diluted ten times with water. When excess is present its degree may be estimated roughly by determining the highest dilution of urine which will produce a reddish tinge.

### Acetone Bodies in the Urine

Hydroxybutyric acid, aceto-acetic acid, and acetone may all appear in the urine in the condition known as ketosis. This occurs in certain cases of uncontrolled diabetes and in starvation.

**Test for acetone bodies.** The urine must be fresh and unboiled, as aceto-acetic acid readily decomposes. Rothera's *nitro-prusside test* is performed first, as follows:

Ten ml. of the urine are saturated with ammonium sulphate by adding an excess of the crystals; 3 drops of a strong freshly prepared solution of sodium nitro-prusside and 2 ml. of strong ammonia solution are then added. A deep permanganate colour is produced. This test is given both by acetone and aceto-acetic acid but no other substances that may occur in fresh urine.

If Rothera's test is negative, acetone bodies are absent; if positive

aceto-acetic acid may be tested for by the *ferric chloride reaction* (Gerhardt's test), as follows:

Take some urine in a test-tube, and add 10 per cent ferric chloride solution drop by drop. A precipitate usually forms and disappears again on adding more ferric chloride. The solution becomes brownish-red if aceto-acetic acid is present.

Aspirin, antipyrin, salol, salicylates, phenol, and some other drugs give a similar colour with ferric chloride. Prolonged boiling (before adding the ferric chloride) destroys aceto-acetic acid, but the other substances which give a colour with ferric chloride are unaffected. If, therefore, urine which has been subjected to prolonged boiling still gives the ferric chloride reaction, it may be inferred that the reaction was not due to aceto-acetic acid. Boiling *after* adding ferric chloride destroys the colour, whether this is due to aceto-acetic acid or to other substances.

A positive ferric chloride reaction is obtained only if aceto-acetic acid is present in considerable amount. If, therefore, the urine reacts to Rothera's test but not to ferric chloride, it may be inferred that only small quantities of acetone bodies are present. If both are positive, the patient has a ketosis of considerable severity, demanding urgent treatment.

*Acetest* tablets provide a simple means of testing the urine for ketones. One tablet is placed on a clean white surface and one drop of urine placed on the tablet. If ketones are present, a mauve colour develops. The depth of the mauve colour depends on the concentration of ketone bodies, but the combination of the Rothera and Gerhardt tests is probably more reliable for quantitative purposes.

### III ESTIMATION OF RENAL EFFICIENCY

The basic function of the kidneys is to rid the body of the waste products of metabolism, which are presented to them in the blood-stream. This they do by the excretion of waste products into the urine and by the selective retention of those substances which are not waste. The former is carried out primarily by the glomeruli, which filter off all the contents of the plasma except those of high molecular weight, and the latter by the tubules which concentrate the glomerular filtrate by the removal of water, and alter its composition by the selective re-absorption of its solutes and by the active excretion into it of other substances. Progressive damage to the kidney by chronic renal disease is associated with destruction of or cessation of function in a large number of glomeruli. This

leads to a reduction in the overall glomerular filtration-rate and a rise in the blood urea. Under these conditions there is a much greater flow of urine through the remaining tubules, which overloads the diluting and concentrating mechanisms at the distal ends of the tubules. The result is a progressive diminution in the ability of the kidney to concentrate and dilute urine as renal destruction progresses until finally the specific gravity becomes fixed at 1010.

Numerous tests are available to demonstrate lesser degrees of failure, and depends on ways of measuring excretion of waste products, on observing the response of the kidney to some form of stress or on measuring the rate of excretion of an easily identifiable foreign substance introduced into the body.

Clearance tests are relatively delicate ways of measuring the ability of the kidney to handle excretion products. The renal clearance of a substance is the volume of blood which is completely cleared of it in one minute, and can be calculated if the blood-level and the amount excreted in the urine in a given time is known. Urea and creatinine clearances are commonly used amongst tests of renal function, and since these substances are excreted by the glomeruli and not by the tubules, their clearance depends on glomerular function. Kidney function may be tested by stress in two ways. The ability to concentrate urine is measured by depriving the patient of water for a fixed period and observing the specific gravity of the urine; or by giving him a load of urea and measuring the resulting urinary concentration of this substance. The ability to dilute urine is determined by giving a water load. Such tests largely depend on tubular re-absorption.

The ability to excrete the dye phenol-sulphone-phthalein is another method of testing renal function. This substance is primarily excreted by the tubules, and its rate of excretion depends on this component of renal function as well as on renal blood-flow.

In chronic renal disease with progressive destruction of nephrons, the glomerular filtration rate and concentrating power are reduced *pari-passu*. In 'extra-renal uraemia', due to heart failure or dehydration, the glomerular filtration may be diminished but the concentrating-power may be normal.

#### **The Phenol-Sulphone-Phthalein Test for Renal Efficiency**

The principle of the test is to estimate the amount of the dye phenol-sulphone-phthalein excreted in two hours after the injection of 6 mg.

In order to secure a good flow of urine, 400 ml. of water are given to drink. After about 15 minutes, the patient's bladder is emptied, if necessary by catheter, and 6 mg. of the dye dissolved in 1 ml. of water are injected intramuscularly or intravenously.

After exactly one hour, and again after two hours, the bladder is emptied, and the whole of these two specimens reserved for estimation of the dye colorimetrically in each.

The amount of the original dose excreted in the first hour by a normal adult is usually over 40 per cent, and in two hours together over 60 per cent.

#### Urea-Concentration Test

The patient is given a test dose of urea, which should, if renal efficiency is unimpaired, provoke a high concentration in the urine.

**Method.** 15 g. of urea in 100 ml. of water flavoured with a little tincture of orange is given by the mouth, when the patient has had nothing to drink for nine hours, and after emptying the bladder; the urine passed in each of the subsequent three hours is collected and the urea-concentration estimated preferably in the specimens from the second and third hours.

If the kidneys are healthy, the concentration in the second and third hours is usually over 2·5 per cent and almost invariably over 2·0 per cent unless the volume is large (over 150 ml. per hour). With moderate damage to the kidney, concentrations from 1·5 to 2·5 per cent may be encountered, and with severe damage under 1·5 per cent. A certain amount of judgement is required in interpreting the result; such high concentrations cannot be expected when the initial blood-urea is low (e.g. about 0·020 per cent) as when it is comparatively high (e.g. about 0·045 per cent); nor such high concentrations when the volume passed is comparatively large (e.g. 130 ml.) as when it is small (e.g. 50 ml.).

This test is a useful complement to the phenol-sulphone-phthalein test, as the latter sometimes gives low figures when the kidneys are little affected. If low figures for phenol-sulphone-phthalein are accompanied by low figures for urea-concentration, there is little doubt that the kidneys are deficient. On the other hand, the phenol-sulphone-phthalein test rarely gives normal figures when the renal efficiency is reduced, and high figures for phenol-sulphone-phthalein confirm the urea-concentration test in its doubtful zone between 2·0 and 2·5 per cent.

#### IV MICROSCOPICAL EXAMINATION OF URINARY DEPOSITS

The urine is centrifuged and a drop of the deposit is placed on the

centre of a slide and covered with a cover-glass. Preparations may also be made by allowing the urine to stand in a conical glass and removing some of the deposit at the bottom of the glass with a pipette. The preparation is then examined with both the low ( $\frac{1}{2}$  in. or  $\frac{2}{3}$  in.) and high ( $\frac{1}{2}$  in.) objectives, the microscope being vertical and the diaphragm partly closed.

**1 Red blood corpuscles** (Plate IX) are best sought in recently voided urine. Usually of normal size, they may, according to the density of the urine, appear either swollen or shrunken and crenated. They are recognized by their shape and biconcavity, and particularly by their colour. Droplets of oil from a catheter are frequently mistaken for red blood-corpuscles. The droplets are readily differentiated by their higher refractive index, more circular shape, and variable size. The presence of small numbers of red cells in the urine—so-called 'microscopic haematuria', may cause no change in its macroscopic appearance and give no positive guaiac reaction. Its detection, which can only be achieved with the aid of the microscope, may be of great importance in diagnosis, particularly in cases of subacute bacterial endocarditis. An occasional red cell may be found in apparently normal urine. More than one per ten high-power fields should be regarded as abnormal.

**2. Leucocytes or pus cells** (Plate IX) can generally be recognized by their round shape, the lobed form of their nuclei and their refractile granular appearance. When numerous, they may appear in clumps or groups. Pus may form a deposit visible to the naked eye in the specimen glass, and may give a green colour with the guaiac reaction, but a certain diagnosis can only be made by seeing pus cells under the microscope. An occasional leucocyte may be seen in the deposit from the urine of apparently normal persons, but the presence of pus cells in any number is always pathological. One must make sure that the urine has not been contaminated, and in women a catheter specimen is essential if the urine is to be examined for either blood or pus.

**3 Epithelial cells** (Plate IX) from various parts of the urinary tract may be found in the urine.

**4 Spermatozoa** occur at times in the urine, where their characteristic appearance makes it easy to recognize them.

**5 Prostatic threads** are found when there is chronic inflammation of the prostate, especially after gonorrhoea. They are much larger than tube-casts, being visible readily enough to the naked eye as they float in the urine or on its surface.

**6 Tube-casts.** The recognition of casts (Plate IX) is important as their presence in any number usually indicates that the patient is suffering from some form of nephritis. They are first recognizable under the  $\frac{1}{2}$  objective, but should be further examined under the  $\frac{1}{4}$ . Casts are distinguished from other objects which may be mistaken for them—such as hairs, wool, cotton, masses of urates, prostatic threads, rolled-up epithelial cells, and so-called 'cylindroids'—by their shape and sharply defined outline. They are always cylindrical in shape and may have rounded ends, or one end may be ragged as if fractured. Granular casts contain fine or coarse granules. Hyaline casts are pale, transparent, and homogenous. An occasional hyaline cast may be found in apparently normal urine: granular or epithelial casts or hyaline casts in numbers usually indicate a nephritis of some variety, though they may also be seen in right heart failure and in septicaemias.

Other varieties of casts which are described, include those formed from red blood-corpuscles, leucocytes, epithelial cells, colloid substances, and lipoid materials. Cylindroids, which resemble tube casts but are extremely long and narrow and usually tapered, flattened, or frayed at the ends, are of no importance.

**7 Tumours of the bladder.** These, especially when villous, may very rarely be detected by the presence of *fragments of the growth* in the urine.

**8 Bilharzia.** *Bilharzia haematobium* is the only *parasite* of any importance found in the urine. The ova (Plate XII: 19) measure 0·12 mm. by 0·44 mm. A spine projects at one pole. The ova of *Bilharzia mansoni*, which are found in the faeces, have a lateral spine.

## V OTHER METHODS

**Intravenous Pyelography.** This depends on the excretion by the kidney of certain radio-opaque organic compounds of iodine. These are injected intravenously, and X-ray films of the kidney are then taken after various time intervals. If the kidneys are functioning adequately, the renal pelvis, ureters, and bladder are thus outlined.

This investigation is of value in the diagnosis of hydronephrosis, tumours of the kidney, polycystic disease, and in the detection of a non-functioning kidney.

**Cystoscopy.** The interior of the bladder may be inspected through a cystoscope, which is inserted *via* the urethra. The main value of this investigation is in the diagnosis of tumours of the bladder. It may also be used to insert fine catheters into the ureters and thus enable urine from each side to be sampled; or, by the injection of a radio-opaque solution, allow X-rays films to be taken of the pelvis or pelves when renal function is inadequate for intravenous pyelography.

**Renal biopsy.** By means of a special needle inserted into the back, biopsy specimens of the kidney may be obtained. Considerable skill is required, and there is a small but definite risk to the patient. At present the method has little application in routine clinical medicine, although it may, on occasion, be of value in accurate assessment of nephritis from the point of view of steroid therapy, or for the diagnosis of rare conditions such as renal amyloid disease or sarcoidosis of the kidney.

## 8 THE SKIN

For the examination of the skin and its appendages, the patient should be stripped as completely as circumstances permit, and should be examined by daylight.

### Colour

First notice the colour of the skin. The normal colour is very variable, some persons having a fresh complexion, and others, though quite healthy, a pale one. Pallor is also often seen in a variety of illnesses. It may be seen temporarily in haemorrhage, shock, and intense emotion. Anaemic persons are often pale, but not all pale persons are anaemic. The colour of the mucous membrane of the eyelids and mouth is a better indication of anaemia than is the colour of the skin. *Undue redness* is seen in overheating, extreme exertion, sunburn, some fevers, and in many of the exanthemata. *Cyanosis* is a bluish or purplish tint, which may be more or less generalized or limited to one or more extremities. It is due to the presence of an excess of reduced haemoglobin resulting from impaired oxygenation or circulation of the blood (p. 23). It is important to note that *methaemoglobinaemia* may produce a blue tint which is less bright and more leaden than cyanosis. Methaemoglobinaemia may be due to poisoning by aniline, nitro-benzene, or drugs such as phenacetin or sulphamilamide.

*Jaundice* varies from the 'sub-icteric', 'lemon-yellow', or 'daffodil' tints seen in pernicious anaemia and acholuric jaundice, to various shades of yellow, orange, or dark olive-green in obstructive jaundice. Jaundice must be distinguished from yellowness due to the taking of mepacrine, either as a prophylactic against or treatment for malaria in those who have recently returned from the tropics, and from rare cases of 'carotinaemia', due to the presence of an excess of lipid-soluble yellow pigments in the plasma. Neither mepacrine nor carotene, however, stain the conjunctivae, which jaundice does. Slight degrees of jaundice cannot be seen in artificial light.

Normal skin contains varying amounts of brown *pigment*. A congenital absence of pigment in the skin, which may be generalized or occur in patches, is known as *albinism*. Alternating patches of white and darkly pigmented skin are seen in *leucomelanodermia*. Increased pigmentation may be racial, due to sunburn, or connected with various diseases. In *Addison's disease* there is a brown or dark-brown pigmentation, affecting exposed parts, parts pressed on by corsets, parts normally pigmented such as the axillae and, very characteristically, the mucous membranes. That of the lips and mouth should always be examined and may exhibit dark bluish-black areas that have been compared with the stains produced by sucking a pen. More or less generalized pigmentation may also be seen in *haemochromatosis*, where it has a peculiar bronze colour with a metallic sheen; in *chronic arsenic poisoning*, where it is finely dappled, and affects covered more than exposed parts; in *argyria*, where the deposition of silver in the skin produces a diffuse slaty-grey hue; and occasionally in the cachexia of advanced malignant disease. More localized pigmentation is seen in *pregnancy*, where it particularly affects the nipples and their areolae and the linea alba; in *hyperthyroidism*, *pellagra*, *rheumatoid arthritis*, and a variety of prolonged wasting diseases. Localized pigmentation is seen in scars of various kinds, particularly those due to X-irradiation therapy and those following varicose ulcers of the legs. *Phtheiriasis* or *vagabond's disease*, a pigmentation due to chronic infestation with lice, is now rarely seen. *Erythema ab igne*, a coarsely mottled pigmentation of the legs of women who habitually sit too near a fire, is, however, common (Plate III).

### Haemorrhage

Haemorrhages into the skin occur in various forms and in various conditions. If less than 1 mm. in diameter they should be referred to as *petechiae*; if from 2 to 5 mm. in diameter as *purpuric spots*, and if larger as *echymoses*. If the haemorrhage is large enough to produce an elevation of the skin, it is referred to as a *haematoma*.

Petechiae and purpuric spots do not disappear when they are pressed on by a glass slide or lens, which serves at once to distinguish them from erythematous spots, such as the rose spots of typhoid fever, and from *telangiectases*, which consist of a small collection of dilated skin vessels. They must also not be confused with capillary naevi (de Morgan's spots), which are common and have no pathological significance.

### Eruptions

Next, one should seek the presence of any eruption. If present, inquiry should be made on the lines laid down on p. 8. The exact situation and extent of the eruption should be noted, and whether it is symmetrical or confined to one side only. One should then pass to a description of the minute characters of the eruption. In order to do this, it must be remembered that every cutaneous eruption consists of a primary lesion, to which secondary lesions may or may not be superadded.

#### Primary Lesions

**1 Macules (spots).** Any abnormal change in the colour of the skin confined to a limited area. Always note whether or not they fade on pressure. The rose spots of typhoid fever, for example, fade on pressure, whilst those due to haemorrhages into the skin do not.

**2 Papules.** Solid projections above the surface, which are not larger than a pea. The term *tubercle* or *nodule* is applied to any solid projection from the skin which is larger than a pea, but not larger than a cherry. Anything larger than that is called a *tumour*. Always note whether the top of the papule is rounded, as in some forms of eczema, pointed, as in acne, or flattened, as in lichen. As regards the base, observe whether it infiltrates the skin widely or not. The wider the infiltration, the more extensive and severe the inflammation.

**3 Vesicles.** Elevations of the horny layer of epidermis by transparent or milky fluid, which are not larger than a pea. If larger than this, they should be described as *bullae* or *blebs*. Always note whether or not there is an area of redness around the base of a vesicle, for such redness indicates that the vesicle is planted upon an inflamed base—a fact which may be of diagnostic value.

**4 Pustules.** Small elevations of the skin containing pus. Observe whether there is much infiltration around them or not.

**5 Weals.** Slightly elevated portions of skin, the centre of which is paler than the periphery.

Having stated which of these primary lesions it is that composes the eruption, one should next note whether the lesions are isolated

(discrete), or whether they run together (confluent). It must also be remembered that an eruption may be made up of more than one kind of primary lesion. Thus, papules may be mingled with pustules, or pustules with vesicles, and so on.

### Secondary Lesions

Next look for *secondary lesions*. These are either produced mechanically, or are the result of changes which take place in the primary lesion in the course of its growth or decline. The commonest secondary lesions of mechanical production are *excoriations* due to scratching, and *fissures* (rhagades)—deep cracks going down to or into the corium, and produced by the stretching of the skin after it has become inelastic owing to thickening of any kind. Fissures are often very painful.

The following are the secondary lesions produced by changes in those which are primary:

**1 Desquamation.** If the primary lesion is a dry one (macules or papules), a scaling off of epidermic cells occurs, and the eruption is then said to be '*scaly*'.

In moist lesions (vesicles, pustules, bullae) the epidermic cells become glued together by the dried fluid, and a *scab* or *crust* forms, The scab may be serous, purulent, haemorrhagic, or sebaceous according to the nature of the contents of the primary lesions.

**2 Infiltration** may occur around the primary lesions, leading to a leathery feeling in the skin. This is usually the result of prolonged chronic inflammation.

**3 Pigmentation** may occur around the primary lesions. This also is usually due to prolonged inflammation.

**4 Ulceration.** Caused by breaking-down of the primary lesions and destruction of a part of the true skin.

The points to note in describing an ulcer are (i) the nature of the floor of the ulcer and the granulations covering it; (ii) the character of the edge—smooth, raised, undermined, etc.; (iii) the discharge, whether serous, purulent, watery, fetid, etc.; (iv) the character of the surrounding skin, whether indurated, pigmented, etc. It is also important to examine the lymph-nodes that drain the area of the ulcer.

**5 Scar-formation.** This only occurs where the true skin has been involved, i.e. where there has been an ulcer or an equivalent injury. Describe the scar, noting especially whether it is thin or thick, freely movable or adherent to the deeper tissues, pale or livid, pitted or not, surrounded by a zone of pigmentation or not.

### Palpation of the Skin

Proceed now to the palpation of the skin. Pass the hand gently over it, pinching it up between the forefinger and thumb, and note the following points:

Is it smooth or rough, thin or thick, dry or moist? If there is any visible sweating, note whether it is general or local. The elasticity of the skin should be investigated. If a fold of healthy skin is pinched up, it immediately flattens itself out again when released. Sometimes, however, it only does so very slowly, remaining for a considerable time in a creased condition. This may be of little or no significance in old persons with loose inelastic skins, but may be an important sign of dehydration in conditions associated with prolonged vomiting and diarrhoea.

The conditions of the subcutaneous tissue should be investigated. The presence of *oedema* is usually recognized by the fact that if the skin is pressed with the finger, especially over a hard body such as a bone, a pit is left which persists for some little time. In some cases, no pitting can be produced, especially when the oedema is of very long standing. The best place to look for slight degrees of oedema in cardiac disease is behind the malleoli of the tibia and fibula in patients who are ambulant, and over the sacrum in those who are confined to bed. The pressure of the finger should be maintained for 20 to 30 seconds, or small degrees of oedema will be overlooked. Pitting is absent in oedema, owing to lymphatic obstruction, where the skin is usually thickened and tough.

*Subcutaneous emphysema* gives rise on palpation to a characteristic crackling sensation. It starts in, and is usually confined to, the neighbourhood of the air-passages or air-containing organs. In rare cases it may be due to infection with gas-gangrene organisms.

### Microscopical Examination

Microscopical examination of the skin and its appendages is useful in the diagnosis of some *parasitic diseases*, of which the following are the chief:

**1 Scabies or itch.** This is due to the *Acarus (Sarcoptes) scabiei*. The female acarus is larger than the male, and forms burrows in the skin, in which the eggs are deposited. These burrows should be looked for between the fingers and on the inner aspects of the wrists. They are recognized with the naked eye as little short dark lines terminating in a sort of shining spot of skin. The eggs lie in the dark line, the insect in the shining spot. It may be picked out by means of a flat surgical needle passed along the black line to the clear spot. The use of a lens aids the operation—which is by no means invariably successful—and makes possible the recognition of the insect. The latter may be placed on a slide under the microscope for more detailed examination.

**2 Pediculosis.** Three varieties of pediculus occur—*Pediculus capitis* on the head, *P. corporis* on the trunk, *P. pubis* on the pubic and axillary hairs. The eggs or 'nits' of *P. capitis* are stuck on the hairs. From their position on the hairs one can judge roughly of the duration of the condition, for they are fixed at first near the root of the hair, and are then carried up with the latter in its growth. The higher up the nits are, therefore, the longer the pediculi have been present. *P. corporis* should be looked for in the seams of the clothes, especially where the latter come into close contact with the skin—e.g. over the shoulders. The bites of the parasite produce haemorrhagic spots, each with a dark centre and a paler areola. Marks of scratching should always be looked for on parts accessible to the patient's nails.

*P. corporis* is the longest of the three, *P. pubis* is shortest, and *P. capitis* is between the two in size. *P. pubis* is also distinguished from the others by being yellowish-brown in colour. *P. capitis* and *P. corporis* are both greyish in colour, though the latter varies considerably with the colour of the skin of its host. The shape of the thorax and abdomen forms a distinguishing character between these varieties and *P. pubis*.

**3 Fungus infections.** Fungus may grow in the skin, nails, or hair and cause disease (ringworm). *Skin.*—Between the toes, on the soles of the feet and in the groins are the commonest sites. The lesions may be scaly and vesicular areas tending to spread in a ring form and healing in the centre; scaly erythematous plaques with festooned margins; areas of hyperkeratosis on the parts of the skin which have a thick horny layer (palms and soles); or macerated, dead-white offensive-smelling epithelium in the intertriginous

areas such as the toe clefs. *Nails*.—Discoloration, deformity, hypertrophy, and abnormal brittleness may result from fungus infection. *Hair*.—Ringworm of the scalp is most common in children. It presents as round or oval areas of baldness covered with short, broken-off, lustreless hair stumps. These hair stumps usually give a bright green fluorescence when exposed to long-wave ultraviolet light (UVL filtered through Wood's glass).

### Microscopical Examination

Scales from the active edge of a lesion are scraped off lightly with a scalpel, or the roofs of vesicles are snipped off with scissors. The material is placed in a drop of 10–20 per cent aqueous potassium hydroxide solution on a microscope slide, covered with a cover-slip and left for 30 minutes to clear. It is then examined with  $\frac{1}{2}$ -in. or  $\frac{1}{6}$ -in. objective, using low illumination. The mycelium is recognized as branching refractile threads which boldly transgress the outlines of the squamous cells (mycelium which respects the cells' outlines is 'mosaic fungus', an appearance probably produced by intercellular lipoid). Nails are examined in much the same way, but as nail is harder and denser it is necessary to break up the snippings and shavings into small fragments. These are either heated in potassium hydroxide or are left to clear in it overnight before being examined. A scalp lesion is cleaned with 70 per cent alcohol or with 1 per cent cetrimide, and infected stumps are extracted by traction in the long axis of the hair with epilation forceps (this is most conveniently done under Wood's light). The hairs are cleaned in potassium hydroxide in the same way as skin-scales. Examination under the microscope reveals spores on the outside of the hair roots, and mycelium inside the hair substance. The species of fungus responsible may be established by culture on Sabouraud's glucose-agar, or on beerwort-agar medium.

## **9 THE NERVOUS SYSTEM**

### **General Considerations**

The aim of a neurological examination is to determine the site and nature of the lesion responsible for the patient's symptoms in a case of nervous disease. It is also an essential part of any routine examination, for signs may be found in the nervous system in the absence of symptoms, which lead to the diagnosis of conditions affecting other parts of the body. Precise history-taking is also important, as the evolution of any particular condition must be known if an accurate diagnosis is to be achieved. Due regard should be paid to the previous and family histories, which may yield valuable clues to the nature of the patient's illness, and the examination of other systems must not be neglected; for instance, inspection of the skin, skeletal system, chest, abdomen, or cardiovascular system may provide information leading to the diagnosis of a nervous disorder.

A detailed neurological examination is an ordeal for ill patients and a test of concentration and co-operation in those in good general health. Care should be taken not to fatigue the patient unduly. Overlong examination may defeat its own ends, especially when sensation is being investigated, by leading to variable and incongruous findings. It may be necessary to conduct the examination in more than one session.

Whilst the mode of examination outlined in the pages which follow is that generally adopted, it need not be rigidly adhered to. For example, if a patient is complaining of sciatic pain, there is no reason why one should not begin with the examination of the lower limbs and lumbar spine.

Observation of the patient's ordinary activity, for example the way he walks into the room and undresses for examination, is often helpful. The minimum record of a negative neurological examination should include a statement that the optic discs, pupils, other cranial nerves, and motor and sensory systems are normal; a record should

be made of the state of the tendon reflexes and of the abdominal and plantar responses.

## I ANATOMY AND PHYSIOLOGY

The *dominant hemisphere* is that which plays the major part in control of a person's activities, especially that of speech, and is situated on the left side in right-handed people and on the right in left-handed people.

### The Motor System

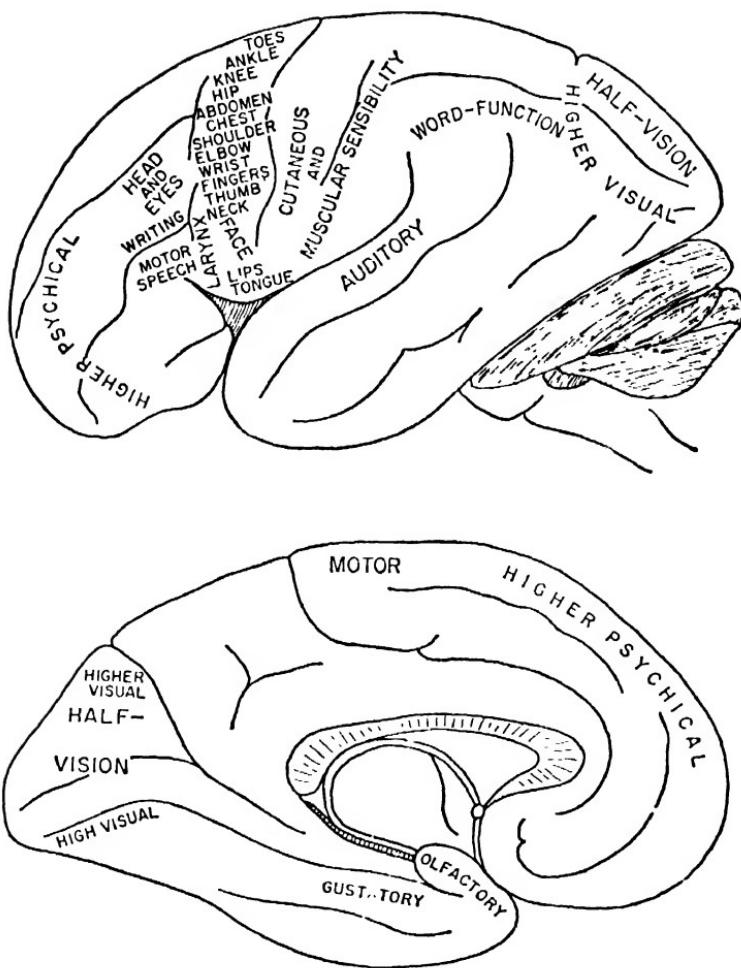
#### The Lower Motor Neurone

Muscular movement depends ultimately on the integrity of the lower motor neurones, which connect striped muscles with the central nervous system. The lower motor neurones consist of the anterior horn cells and homologous cells in the brain-stem, and their fibres, which pass *via* the anterior spinal nerve roots and peripheral nerves to the muscles. The nerve impulses which influence the muscles pass *via* this final common pathway to the motor end-plates and are then transmitted humorally to the muscle fibres. If this pathway is interrupted at any point, muscular wasting and weakness, hypotonia and loss of tendon reflexes occur. These are the cardinal signs of a lower motor neurone lesion.

Although various reflex movements operate at a spinal level, the initiation of voluntary and more complex movements and the maintenance of posture and muscle tone depend on impulses arising from higher centres. These impulses can only reach the muscles if the final common path is intact. These higher centres consist of the pyramidal and extra-pyramidal systems and the cerebellum.

#### The Pyramidal System

This system consists of the pathways which link the cerebral cortex directly with the lower motor neurones in the brain-stem and spinal cord. The fibres concerned are gathered in the pyramidal tracts. They arise from the motor area of the brain, but the pyramidal tracts also contain fibres which arise from the post-central cortex and subcortical structures. The motor area of the brain occupies the anterior wall of the fissure of Rolando and the adjacent parts of the pre-central gyrus. There is localization of function in the motor cortex, different parts of the opposite side of the body being separately represented. Those parts of the body which carry out the most skilled movements, for example the fingers and thumb, have the largest areas of repre-



**Fig. 47.** The functional areas of the left hemisphere showing outer (above) and mesial aspects (below).

sentation. The areas for the tongue, jaw, and facial movements lie lowest in the motor cortex, those for the arm, trunk, and leg following successively as the motor area ascends on to the medial aspect of the hemisphere (Fig. 47 and 48).

The fibres of the pyramidal tracts pass downward from their

## THE NERVOUS SYSTEM

cells of origin into the internal capsule, occupying the anterior two-thirds of the posterior limb (Fig. 50). Here the order of representation of the body is face, shoulder, elbow, hand, trunk, and lower limb from before backwards. The pyramidal fibres now descend to occupy the middle three-fifths of the peduncles of the mid-brain in the same order; below this level there is no segregation of the fibres according to their destination, consequently a lesion affecting the tract does not produce paralysis of a single limb. Passing through the

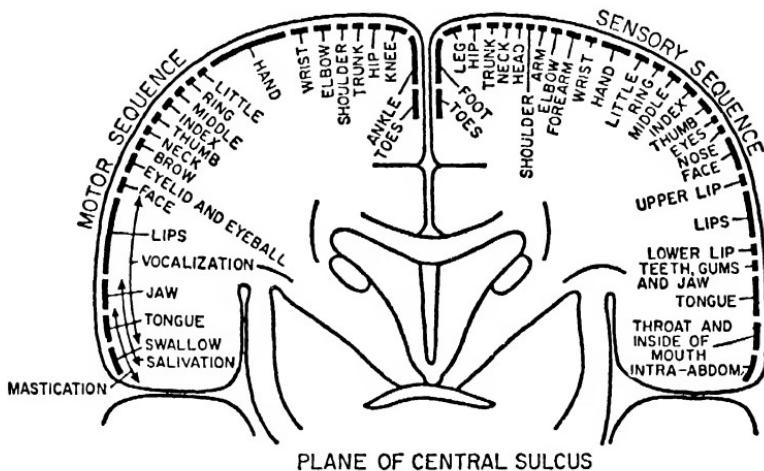
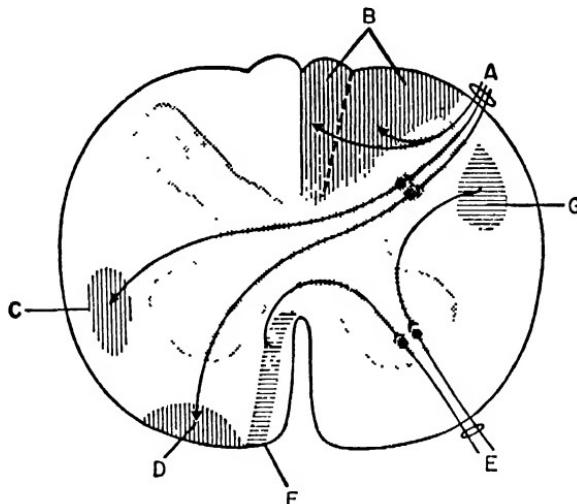


Fig. 48. Rasmussen and Penfield's diagram of localization in motor (left) and sensory (right) cortex.

pons, the tract becomes broken into scattered bundles by the transverse pontine fibres and nuclei pontis. In the upper part of the medulla the fibres join to form the pyramids, which are well marked protuberances on the anterior aspect of the brain-stem. In the lower part of the medulla the greater number of fibres decussate with those of the opposite side and pass backwards, to run down the spinal cord in the lateral columns as the crossed pyramidal tracts. A smaller number of fibres do not decussate, continuing downwards in the anterior columns as the direct pyramidal tracts, but these eventually decussate at lower levels in the anterior commissure. Some uncrossed fibres descend in the crossed pyramidal tract of the same side, ending in the anterior horns of the same side. All the pyramidal

fibres terminate at different levels in the grey matter of the brain-stem or spinal cord, usually ending in relation with internuncial cells rather than with the anterior horn cells themselves.

The pyramidal system is concerned with the initiation of the more voluntary and skilled motor acts. The paralysis resulting from lesions of the system is usually widespread, as of one side of the body



**Fig. 49. Situation of sensory and pyramidal tracts in the spinal cord.**

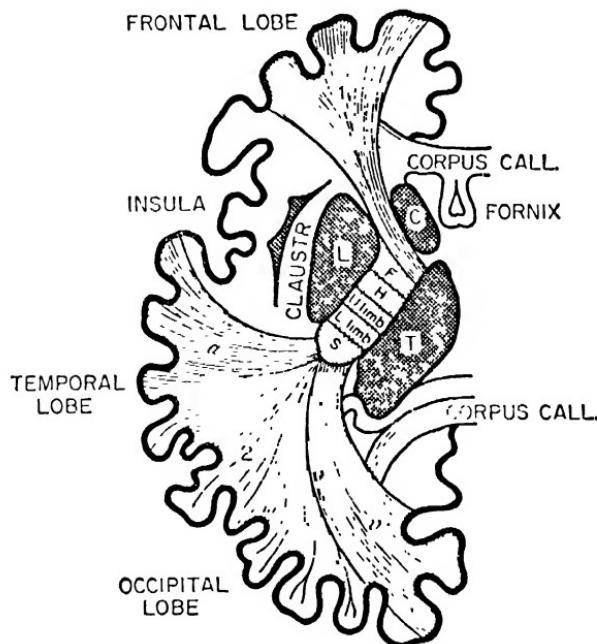
- A. Posterior root (sensory)
- B. Posterior columns (muscle sense, vibration, touch)
- C. Lateral spino-thalamic tract (pain, temperature)
- D. Anterior spino-thalamic tract (touch)
- E. Anterior root (motor)
- F. Anterior cortico-spinal tract (uncrossed pyramidal fibres)
- G. Lateral cortico-spinal tract (crossed pyramidal fibres)

(hemiplegia) or of a whole limb (monoplegia). In hemiplegia due to pyramidal disease, the movements of the head and trunk which are bilaterally innervated often escape altogether. Besides causing weakness or paralysis of movements, pyramidal lesions produce increased muscular tone and exaggeration of the tendon reflexes.

When the pyramidal system is suddenly damaged or destroyed, as by haemorrhage or injury, there is a temporary depressant effect on the lower motor neurones. The resulting paralysis is accompanied by loss of muscle tone and abolition of tendon reflexes. If the patient survives, the characteristic hypertonia and increased reflexes of a pyramidal lesion appear after a time.

### The Extra-Pyramidal System

This is a term applied to the higher centres in the nervous system, excluding the motor cortex and pyramidal tracts, which are concerned with movement and posture. The system includes the basal ganglia, the subthalamic nuclei, the substantia nigra, the red nuclei, and other



**Fig. 50. Internal capsule and corona radiata.**

T, Thalamus; L, Lenticular nucleus, C, Caudate nucleus; F, Supranuclear tract for the facial nerve; H, Supranuclear tract for the hypoglossal nerve; U, Limb, Supranuclear tract for arm muscles, L, limb, Supranuclear tract for leg muscles; S, Sensory tracts (thalamo-cortical tracts); a, Auditory tracts to temporal lobe; v, Visual tract to occipital lobe; 1, Fronto-pontine tract and corona radiata; 2, Occipito-temporo-pontine tract and corona radiata.  
(After Bing.)

structures in the brain-stem. The connections of these extra-pyramidal centres are imperfectly understood, but they include fibres from the cerebral cortex and the thalamus. As far as is known, there are no direct pathways from the basal ganglia to the spinal cord; the connections with the lower motor neurones are indirect, *via* several

paths arising in the brain-stem; these include the rubro-spinal, reticulo-spinal, vestibulo-spinal and olivo-spinal tracts.

Little detail is known of the functions of the extra-pyramidal system. It is concerned with the regulation of muscle-tone, and

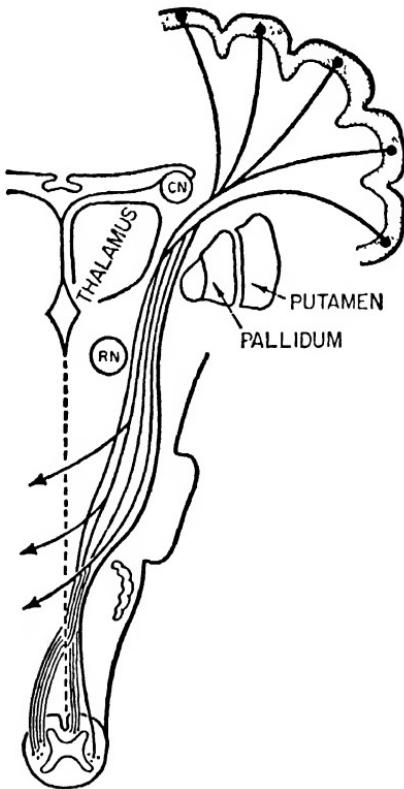


Fig. 51. Diagram of the course of the pyramidal tract.

appears to play a part in the production of the more automatic bodily movements. Diseases affecting the extra-pyramidal system are characterized by alterations in muscle-tone and the appearance of involuntary movements. Muscular power is rarely weakened, but movement is often slowed.

### The Cerebellum

The cerebellum receives afferent fibres from the spinal cord,

vestibular system and cerebral cortex. It influences the lower motor-neurone mainly through its connections, *via* the thalamus, with the basal ganglia and cerebral cortex. It regulates the range, rate, rhythm and force of muscular contractions. Lesions of the cerebellum cause muscular hypotonia and inco-ordination (motor ataxia). No paralysis results, although there may be fatigability of muscles. The inferior vermis is especially concerned with equilibration, and disease affecting this part produces trunk ataxia. Patients thus affected have difficulty in standing erect, and tend to fall backwards or, less frequently, forwards.

### The Sensory System

Sensory impulses from the periphery are conducted to the spinal cord by the afferent nerves, through the posterior root ganglia and the posterior spinal roots. These constitute the first sensory neurones. But only a small proportion of these impulses ever reach consciousness as sensations. The rest are concerned in spinal-reflex functions, in the maintenance of the tone of muscles; or they terminate in those portions of the spinal cord, or in higher centres such as the cerebellum, which control the co-ordination of muscular activity.

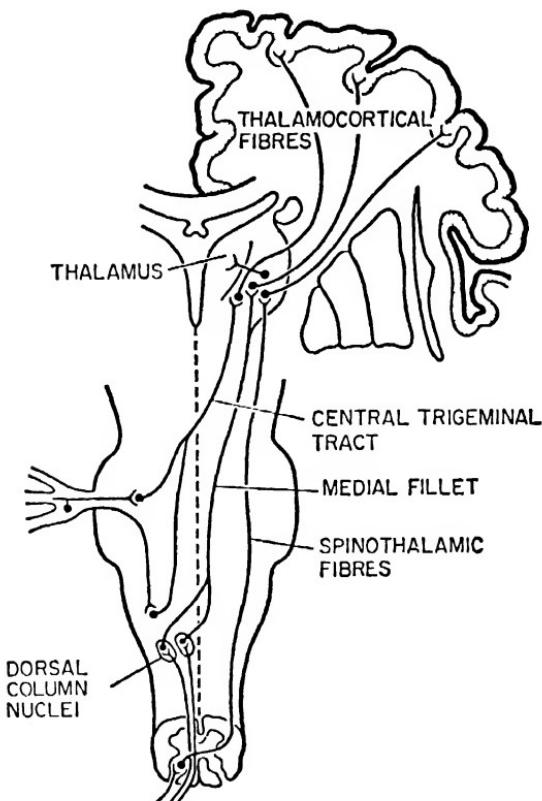
Sensory impressions come not only from the skin and superficial tissues, but also from muscles, tendons, and joints. Disease of the first sensory neurones may consequently affect (i) cutaneous sensibility, and abolish or disturb the perception and localization of tactile, painful and thermal stimuli; or (ii) deep sensibility, which is conveyed by the afferent fibres that come from the muscles, tendons, bones and joints. This system underlies the recognition of position and movement, and, when cutaneous sensibility is lost, heavy touches or the pain produced by pressure can be appreciated through it. Owing to the wide anastomosis and distribution of the fibres of this system, deep sensibility frequently escapes in areas in which cutaneous sensibility is lost; then firm touches, as those produced by a finger or the point of a pencil, may be felt, though light touches cannot be appreciated.

After they have entered the spinal cord, the various sensory impulse channels are rearranged and grouped into other systems. The majority of the peripheral neurones that have carried them higher terminate in the grey matter of the posterior horn at or near the level at which they enter, and from this grey matter the secondary sensory tracts take origin. Some cross immediately, or within a few

segments, to the opposite lateral and anterior columns of the cord, and in it ascend to the brain-stem. Pain and temperature ascend in the *lateral spino-thalamic tract*, the fibres from the lower part of the body being placed laterally, while the secondary fibres of touch pass to the *anterior spino-thalamic tract*. Other peripheral fibres, however, do not terminate in the grey matter of the spinal cord, but run cerebrallywards in the posterior columns of the same side as that on which they entered the cord; these posterior column fibres carry the impulses upon which depend the appreciation of position, of movement, and of size and shape. Vibration sense is conveyed in the posterior column and these contain also a path for touch. The medial of the two posterior columns, the *fasciculus gracilis*, contains fibres originating in the lower part of the body, whereas the lateral, the *fasciculus cuneatus*, carries fibres predominantly from the upper limbs. At any level of the spinal cord, therefore, there are two major groups of sensory fibres conveying sensory information towards the brain: one, in the anterior and lateral columns carrying touch, pain and temperature from the opposite half of the body, and a second, in the posterior column, conveying the appreciation of posture, weight, size, shape and other qualities of sensation from the same side of the body (Fig. 52). A unilateral lesion of the spinal cord, therefore, produces the Brown-Séquard phenomenon, in which pain and thermal sensibility are lost below the level of the lesion on the opposite side of the body, while on the side of the lesion there is, in addition to spastic paralysis, disturbance of the sense of position and of movement, and loss of the recognition of weight, size, shape and vibration. As touch has a double path, one on the same and another on the opposite side of the spinal cord, it is rarely much affected by unilateral spinal lesions.

At the upper end of the spinal cord the posterior-column fibres terminate in the gracile and cuneate nuclei, and the impulses they carry are taken up by secondary sensory fibres, which immediately cross to the opposite side of the medulla in the fillet decussation. Consequently, in the medulla oblongata all sensory impressions are carried in secondary tracts which lie on that side of the nervous system opposite to the half of the body from which they come. But even here all do not run in a single path, for pain and thermal impressions pass through the lateral part of the bulb, while those conducted by the posterior columns enter the medial fillet (Fig. 52). Higher in the brain-stem the two great sensory pathways are joined by the secondary fibres from the nuclei of the sensory cranial nerves.

Finally, the fibres of the fillet and spino-thalamic tract terminate in the thalamus, no secondary sensory fibres passing uninterrupted beyond it, and from this a tertiary system of sensory fibres conveys sensory impressions by way of the internal capsule to the cerebral cortex (Fig. 53).



**Fig. 52. Diagram of the chief sensory paths within the central nervous system.**

The exact extent of the *cerebral cortex* concerned in reception of sensation is still doubtful; it certainly lies mainly in the parietal lobes behind the fissures of Rolando. It seems probable, however, that certain sensory qualities, such as pain, enter consciousness at a subcortical level in the thalamus. The courses of the fibres and the

position of the centres for the special senses are described in the section dealing with the cranial nerves (p. 224). The speech centres and their connections are described at p. 220.

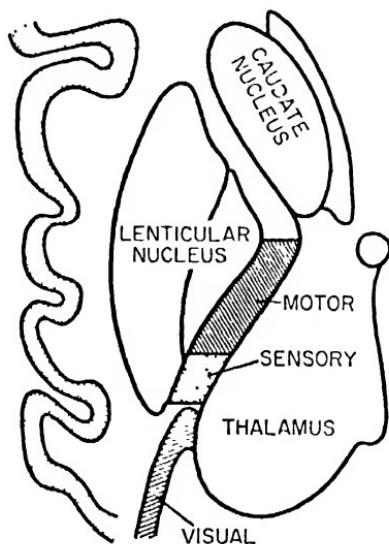


Fig. 53. The internal capsule of the left hemisphere, showing positions occupied by the motor, sensory and visual projection fibres.

### The Spinal Cord

The cord extends as far down as the interspace between the 12th thoracic and 1st lumbar spines; the membranes are continued down as far as the body of the 2nd sacral vertebra.

*The cervical enlargement* reaches to the 7th cervical spine. Its largest part is opposite the disc between the 5th and 6th cervical vertebrae.

*The lumbar segments* lie opposite the 10th and 11th thoracic spines and the next interspinous space.

Physiologically, the cord is to be regarded as made up of a series of superimposed segments, from each of which a pair of nerve-roots arises. To localize focal lesions of the cord, it is necessary to be acquainted with the functions of each segment, and therefore with the area of supply of the pair of nerve-roots arising from it.

Figs. 54 and 55 show the distribution of the cervico-brachial and

## THE NERVOUS SYSTEM

lumbo-sacral plexuses. The sensory functions of the cord are shown in Fig. 56. Detail of the upper cervical segments may be seen in Fig. 62 in relation to the Vth cranial nerve.

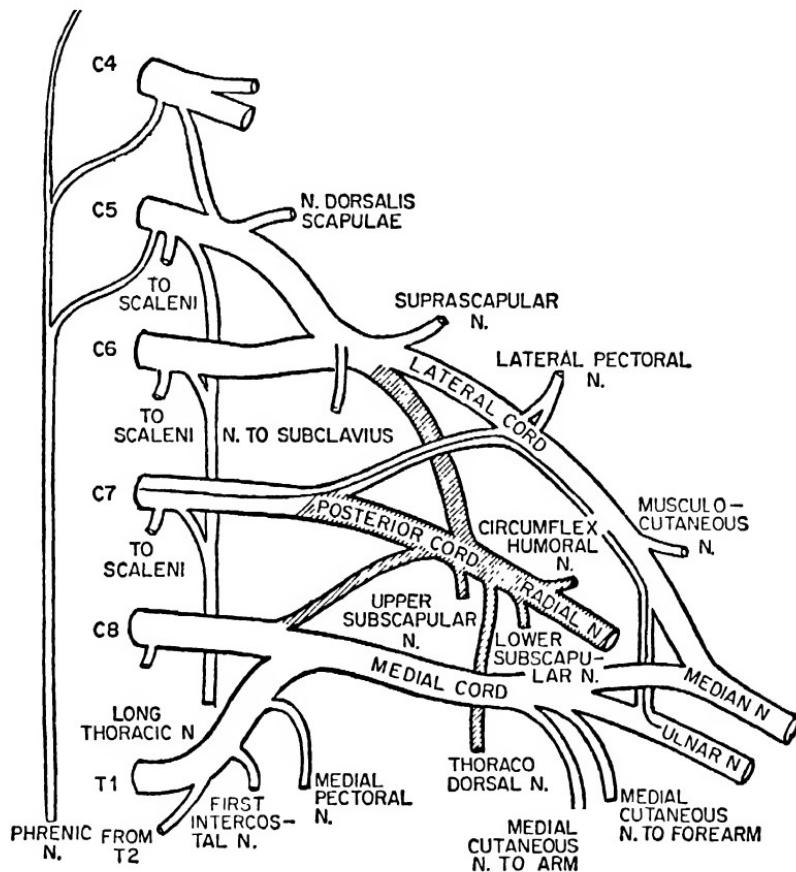


Fig. 54. The Cervico-brachial plexus.

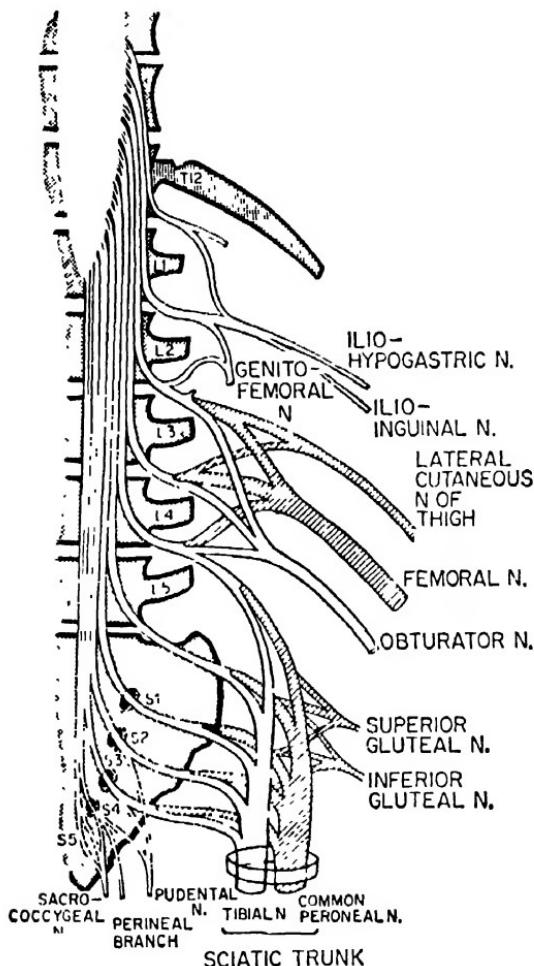
Since the cord ends at the level of the lower border of the first lumbar vertebra, spinal segments evidently do not correspond numerically with the vertebrae overlying them. To determine which spinal segment is related to a given vertebra:

For the cervical vertebrae, add 1.

For dorsal 1 to 6, add 2.

For dorsal 7 to 9, add 3.

The 10th dorsal arch overlies lumbar 1 and 2 segments.  
 The 11th dorsal arch overlies lumbar 3 and 4 segments.  
 The 12th dorsal arch overlies lumbar 5.



**Fig. 55. The lumbo-sacral plexus. Posterior (dorsal) columns are shaded  
 Nerves to single muscles have been omitted.**

CERVICAL SEGMENTS					DORSAL
	5	6	7	8	1
SHOULDER	SUPRASPINATUS				
	TERES MIN.				
	DELTOID	6			
	INFRAСПINATUS				
	SUBSCAPULARIS				
	TERES MAJOR		7		
ARM	BICEPS				
	TRICEPS				
	BRACHIO-RADIALIS				
	SUPINATOR				
	EXTENSOR CARPI RADIAL.				
	PRONATOR TERES				
	FLEXOR CARPI RADIAL.				
	FLEXOR POLLIC. LONG.				
	ABDUCT. POLL. LONG				
	EXTENS. POLL. BREV				
	EXTENS. POLL. LONG.				
	EXTENS. DIGITOR.				
	EXTENS. INDICIS.				
	EXTENS. CARPI ULN				
	EXTENS. DIGITOR. MIN.				
	FLEXOR DIGITOR SUBLIMIS				
	FLEXOR DIGITOR PROFUND				
FOREARM	PRONATOR QUADRAT.				
	FLEX. CARPI. ULN.				
	PALMARIS LONG				
	ABDUCTOR POLL. BREV				
	FLEXOR POLL. BREV.				
	OPPONENS POLL.				
	FLEXOR DIGIT MIN.				
	OPPONENS DIGIT. MIN.				
	ADDUCT. POLL.				
	PALMARIS BREV.				
HAND	ABDUCTOR DIGIT. MIN.				
	LUMBRICALES				
	INTEROSSEI				

Segmental innervation of muscles of the upper limb.

	D12	L1	L2	L3	L4	L5	S1	S2
HIP				ILIO-PSOAS				
THIGH					TENSOR FASCIAE GLUTEUS MEDIUS GLUTEUS MINIMUS QUADRATUS FEMORIS GLUTEUS MAXIMUS OBTURATOR INTERN.			
LEG				SARTORIUS ADDUCT. LONG. QUADRICEPS GRACILIS ADDUCTOR BREVIS OBTURATOR EXT. ADDUCT. MAGN. ADDUCT. MINIM.				
FOOT					SEMITENDINOSUS SEMIMEMBRANOSUS BICEPS FEMORIS TIBIALIS ANT. FXTENS. HALL. LONG EXTENS. DIGIT. LONG. SOLEUS GASTROCNEMIUS PERONEUS LONG. PERONEUS BREV. TIBIALIS POST. FLEXOR DIGITOR. LONG. FLEXOR HALLUC. LONG. EXTENS. HALL. BREV. EXTENS. DIGIT. BREVIS INTRINSIC MUSCLES OF THE FOOT INTEROSSEI			

Segmental innervation of muscles of the lower limb.

The 1st lumbar arch overlies the sacral and coccygeal segments.

It is also necessary to remember that in the lower dorsal region the tip of a spinous process is on a level with the body of the vertebra below.

Fig. 56 (p. 213) shows the sensory distribution of the posterior nerve-roots ('root areas').

Fig. 49 shows the position of the different *tracts of the cord* on transverse section.

The tables on pp. 211 and 212 show the *segmental innervation of the muscles* of the limbs. It may be found convenient for reference in the study of cases of peripheral paralysis. The nerve-supply of the head is considered along with the cranial nerves (p. 224).

The peripheral distribution of some important *sensory nerves* is indicated in Figs. 56 to 58.

**Vascular supply of the brain and spinal cord.** The *brain* is supplied by the internal carotid and vertebral arteries. Owing to the position of origin of the left common carotid, an embolus can enter it more easily than it can the artery of the opposite side. Embolic lesions are therefore more frequent in the left than in the right cerebral hemisphere.

The two *vertebral arteries* unite at the lower border of the pons to form the *basilar*, which runs up the middle of the anterior surface of the pons, and ends by dividing into the two *posterior cerebrals*. It gives off paramedian and short and long circumferential branches which supply the pons and parts of the mid-brain and cerebellum.

The *posterior cerebral* supplies the occipital lobe, the lower part of the temporal lobe, with the uncinate gyrus, the inner part of the crus and the corpus quadrigeminus, and the posterior part of the posterior limb of the internal capsule. Blocking of this artery at its origin will therefore involve the visual centre and the sensory fibres, but thrombosis often involves the calcarine branch and hence the visual centre alone.

The basilar artery supplies the upper surface of the cerebellum; the vertebral supplies its lower surface, as well as the greater part of the medullary oblongata.

The *internal carotid* gives off the *anterior cerebral* artery, which curves round the anterior end of the corpus callosum, and is chiefly distributed to the inner surface of the cerebral hemisphere as far back as the parieto-occipital fissure. It also supplies the superior

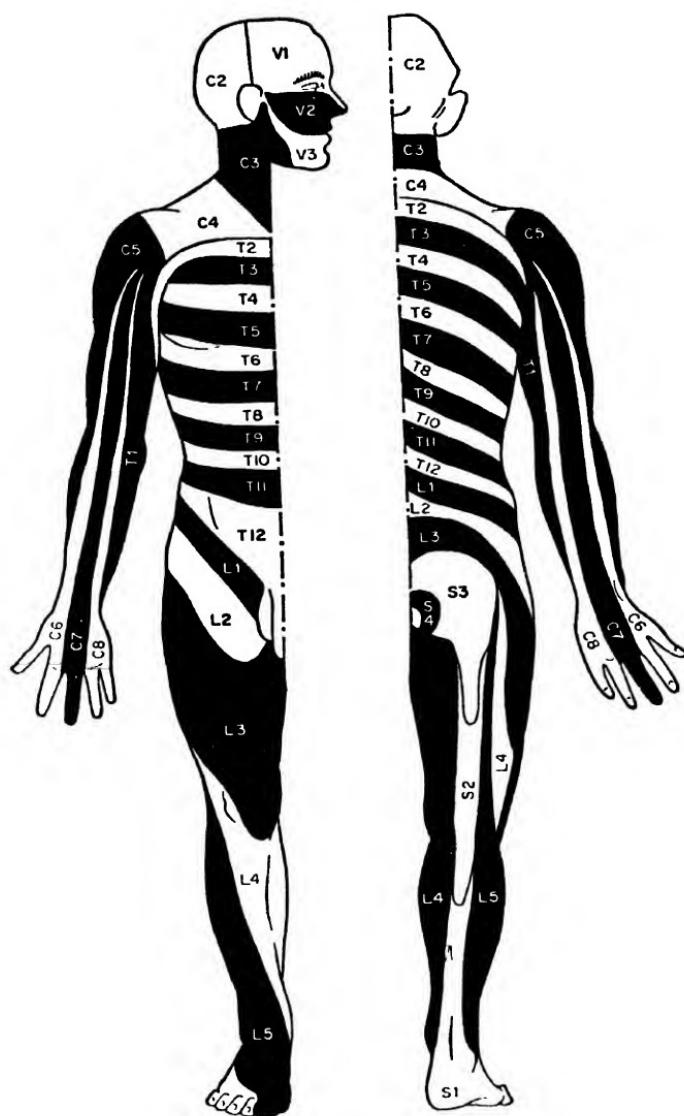


Fig. 56. Diagram of cutaneous areas supplied by sensory roots.  
Minor variations are common.

R—H

frontal convolution, and gives a branch to the anterior part of the internal capsule and to the basal ganglia.

The internal carotid is mostly continued on to the brain as the *middle cerebral*, which lies in the Sylvian fissure. An embolus which has found its way into the internal carotid, therefore, usually ends in the middle cerebral or one of its branches. The middle cerebral gives off *cortical branches*, which supply the motor area and the

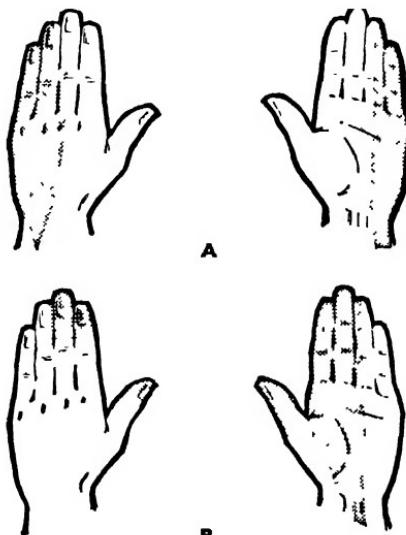
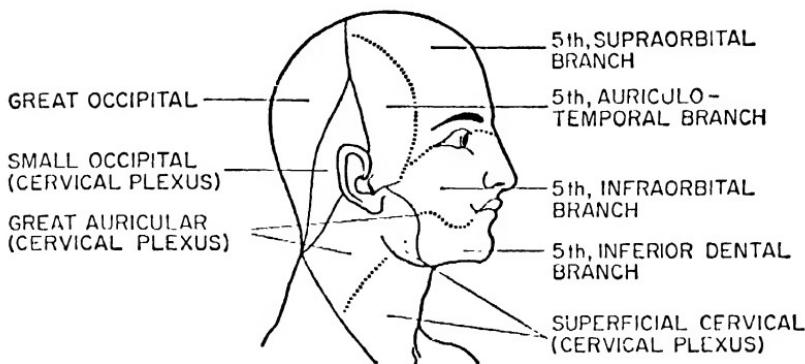


Fig. 57.

A, Area of cutaneous sensory loss after division of the median nerve in the arm. B, Area of cutaneous sensory loss after division of the ulnar above the elbow. These are subject to considerable variation.

upper part of the parietal and temporal lobes. These branches anastomose freely with those of adjoining arteries, hence blocking of one of them may be largely compensated by the establishment of a collateral circulation. It also gives off *central branches* which penetrate into the brain substance and supply the white matter and the basal ganglia. There are two chief groups of these central arteries—an anterior group called the *lenticulo-striate*, and a posterior group, the *lenticulo-optic*. As the lenticulo-striate are more directly exposed to the force of the wave of arterial blood they are more frequently ruptured than are the lenticulo-optic. These central arteries do not

anastomose with one another. They are, therefore, to be regarded as end-arteries. Hence it is that a lesion of one of them is much less likely to be compensated than is a lesion of a cortical branch.



**Fig. 58. Distribution of the sensory nerves of the head.**

Compare with it the segmental distribution as shown in Fig. 62.

The venous blood from the brain is poured into the *venous sinuses*. Owing to the slow current in these, thrombosis readily occurs. The blood from the interior of the lateral ventricles is chiefly returned by the veins of Galen, which end in the straight sinus.

**Spinal arteries.** The *anterior and posterior spinal arteries* arise from the vertebrals and travel downwards in the pia mater, the former in the antero-median fissure and the two latter alongside the posterior nerve-roots. Although they have a long and tortuous course, they do not diminish in size, being reinforced by radicular tributaries from the intercostal and lumbar arteries. The anterior spinal artery supplies most of the spinal cord, only the posterior parts of the posterior horns and columns being supplied by the posterior spinal arteries.

The chief *veins of the spinal cord* are situated dorsally and ventrally in the middle line. Like the arteries, they communicate by radicular branches with the lumbar and intercostal veins, and empty into the vertebral veins. The blood in them flows upwards; hence in compression of the spinal cord, as by tumour or tuberculous abscess, there is venous engorgement below the level of pressure. This results in asphyxia of the spinal cord with paralysis and loss of sensibility,

which frequently disappears if the cause of compression is removed. It also leads to increase in the protein content of the cerebro-spinal fluid below the site of compression, the main feature of Froin's syndrome.

The routine method of examining the nervous system is described in the following pages: The mental state and intellectual functions, including speech, are investigated first. The cranial nerves are tested next, followed by the motor, sensory and reflex functions of the limbs and trunk.

## II INTELLECTUAL AND MENTAL FUNCTIONS

It is important to arrive at some idea of the patient's intellectual state early, as it affords indications that are of help in the subsequent investigation of his symptoms. For example, if his memory is deficient, only a limited value is attached to the account that he gives of the onset of his illness or the state of his previous health. Or if he is comatose, or unable to understand speech, any attempt to investigate the state of his sensory functions is likely to be frustrated. It may be added that it is often essential to obtain a history from the relatives or friends of patients suffering from neurological or psychiatric disorders.

### Appearance and Behaviour

The patient's bearing or his actions when lying in bed are to be noted. Mention whether the patient is unduly disturbed or apathetic, or whether he is in a state of agitation or terror. Is his attention easily held or fleeting? Does he show a reasonable degree of interest in his surroundings? How does he react to the examining doctor's approach and greeting? Is he well-groomed or unkempt? What is the condition of his hair, his beard, his nails, and his hands? Any other feature which strikes one as uncouth in his behaviour (e.g. facial tics, abstraction with his own thoughts) should be noted. Is there any dimming of consciousness, stupor or coma?

Note whether his conversation flows easily or not: whether he is mute, answers only by monosyllables, or is over-talkative. Do his remarks hold together as replies to questions? Do they show looseness of association? Do they show *flight of ideas* (a rushing stream of ideas with some connection) or the 'Knight's move' in association (when one remark follows another with only indirect connection)?

This name derives from chess, in which all pieces move on straight or diagonal lines, except the knight, whose move involves a change of direction. Do his replies to questions suggest *thought blockage*, or does he keep on repeating your question or his own remarks (*perseveration*)? He may use strange words (neologisms) or normal words strung together oddly (word-salad).

### Emotional State

It is important to note his mood. Is he happy or distressed? Is he happier than his condition would warrant (*elation*) or filled with despair or dismay (*depression*)? Does his conversation lead you to feel that there is flattening of emotion, e.g. he speaks of family or financial success without pleasure, or in an incongruous manner (he laughs after relating a misfortune or breaks into tears when given some pleasant news)? Is he able to enjoy anything? Does he feel that nothing is worth while? Does he feel fed up with life, or so fed up that he might as well end it (so that there is a risk of suicide)? Does the play of his features suggest that he enjoys a private world of his own, such as smiling or grimacing at odd times? Does he appear perplexed? Ask him whether persons and things seem as real as they once were, or whether they seem changed in some mysterious way (*depersonalization*). It is, however, unwise to ask leading questions about depersonalization, as neurotic patients will easily 'pick it up'. Note whether he seems irritable or resentful, or whether he receives your words with suspicion.

Inquiries should be made about the patient's *sleep*. Does he sleep too much or too little? If too much, is any particular action likely to precipitate the hypersomnia? If too little, is the difficulty that of falling off to sleep, or of waking frequently, or waking early in the morning and being unable to go to sleep again? Where there is no physical cause for the insomnia, such as pain, cough, asthma, or the wearing of some uncomfortable plaster, the insomnia is likely to be due to some psychological disorder, e.g. the restlessness of mania, the early waking in endogenous depression (melancholia), or the turmoil of the mind with difficulty in getting off to sleep in the reactive depressions (anxiety states).

Inquire about *dreams*. These are frequent and sleep-disturbing in the anxiety states, whether the source of the anxiety is known or unknown. In these conditions the sleep is not refreshing, and a positive complaint of being as tired in the morning as at night may be made.

### **Delusions and Hallucinations**

Delusions are false beliefs which continue to be held despite evidence to the contrary. Hallucinations are false impressions from the organs of special sense (aural, visual, olfactory, etc.) for which no cause is found. The patient's conversation may have already indicated that these are present. Note their content, and the patient's attitude when you express doubt about what seems so real to him.

Neither the delusions nor the hallucinations may be voiced spontaneously. They have then to be jinquired for, and the introduction of the subject may call for considerable diplomacy. Is his mind or his body interfered with by others, or by some physical agency, electricity, wireless, atoms, chemical agencies, poison? Has he felt that others talk about him, or shun him? Are his relatives, neighbours, colleagues at work, kind to him or difficult to get on with? Has he ideas of supernatural power, inordinate wealth, or conversely does he feel that he is weakened, physically, morally, or financially? Has he feelings of guilt? Does he think that he caused his own illness?

Hallucinatory experiences may be carefully hidden. Aural and visual false sense-impressions are commonest. Does he hear or see anything unusual? Does he taste or smell what he has not expected? Hallucinations may only occur at certain parts of the day or in certain places, and this should be noted. Delusions which are secondary to the hallucinatory experiences must be noted, e.g. that 'the neighbours are against him, because his bedroom is filled with a gas, which he assumes the neighbours have engineered'. Notes should be made of any unusual actions upon the patient's part which have been prompted by delusions or hallucinatory ideas, e.g. ideas that one is in the pay of his enemies, or his clutching at small animals which he 'sees' crawling over his bedclothes.

### **Orientation in Place and Time**

Does the patient appreciate his surroundings, and know where he is, or is he wholly ignorant, and does he try to explain his ignorance in some way such as confabulation? Can he tell the date approximately, if not perfectly correctly? Can he tell the approximate time without looking at a watch or clock?

### **Clouding of Consciousness**

The states of clouded consciousness are important to recognize.

*Coma* is a state in which the patient makes no psychologically understandable response to external stimulus or to inner need. In *semi-coma* or *stupor* the patient, although inaccessible, does show some response, for instance, to painful stimuli. Above these deep levels of altered consciousness lie various degrees of confusion, from severe to slight. The questioner must be alert to observe any minor defects in the patient's capacity to grasp what is and what has happened. Such defects will usually be manifest in the responses to tests for orientation, recent memory and appreciation of environment.

### Memory

Inability to grasp and retain images and ideas is a marked feature in acute toxic-delirious reactions and in the subacute and chronic organic psychotic reactions. In these cases recent events may have been registered but cannot be recalled, though it is probably more correct to say that they have not even been registered.

The degree to which recent memory is lost is an index of the degree of organic brain-disorder (not necessarily permanent). Inquire about the day of the week and of the month, the name of the Monarch and of the Prime Minister. Ask the patient to recall what he has read in the paper or heard on the wireless. In framing questions on these lines, regard should be paid to the patient's own particular interests. More incipient changes are discovered by seeing whether the patient can repeat seven digits forwards, or five digits backwards. Bring up a subject discussed three minutes previously and note how much is remembered.

### General Intelligence

It is usually necessary to ascertain the patient's general intelligence, or how it has been affected by brain injury or disease (e.g. arteriosclerosis). The standard which he reached before leaving school, the character of his work and his work record give a rough-and-ready approximation. Frequent changes of job may indicate mental defect or social defectiveness (inability to get on with colleagues). Frequent changes after an accident or a serious illness with a previously good work-record is suggestive of mental impairment.

Tests of memory as given above will indicate the more serious defects, and these can be further exposed by tests of reasoning, more particularly where the tests show inability to criticize. Ask the patient to take sevens from a hundred (i.e. 100, 93, 86, 79 . . .), or to

reverse in his mind's eye the hands of a clock. The absurdities test: 'What would be absurd if I told you I had three brothers, John, Fred, and myself,' etc., indicates grosser disability. A man with relatively low intelligence can give the months of the year parrot fashion, but is unable to say which month precedes May and which October, etc.

### III SPEECH

Proceed next to the investigation of the *speech functions*. In considering speech, it is essential to distinguish between defects of articulation and enunciation, and those disturbances of speech due to diseases of its cerebral mechanism, which we speak of as aphasia.

Supposing that the patient is able to speak, one should note whether there is any peculiarity in his *articulation*. The following are the chief abnormalities which may be present:

#### 1 Stammering

This requires no special description.

#### 2 Lalling, or Baby Speech

Ask the patient to read something aloud. If he lalls, one will recognize that all the difficult consonants are dropped; he speaks like a baby, and, if a child, may perhaps make use of words of his own invention—*idioglossia*. Lalling and idioglossia are usually the result of a congenital defect in the appreciation of the meaning of sounds—congenital auditory imperception.

#### 3 Scanning or Staccato Speech

The patient speaks slowly and deliberately, syllable by syllable, as if scanning a line of poetry. Ask him to say 'artillery'; he will pronounce it 'ar-til-ler-y'. This is the kind of speech found in some cases of disseminated sclerosis.

#### 4 Slurring Speech

The syllables are slurred together as in a state of intoxication. Thus 'British Constitution' becomes 'Brizh Conshishushon.' This kind of speech is met with typically in general paralysis of the insane, and in certain brain-stem disorders.

#### 5 Dysarthria

This disorder of articulation is due to paresis or inco-ordination

of the peripheral mechanism of speech, either of the larynx, tongue or lips, though often all three are affected together.

If the patient's defect consists not so much in a disturbance of articulation as in an ability to produce speech, or to understand it when spoken or when written, then his condition is described as one of *aphasia*.

In order to understand the method of investigating a case of aphasia, it must be remembered that for purposes of speech we require a receptor system involving interpretation of auditory and visual information in word form, the formulation of thought in word form, and its translation into spoken or written speech. A disorder of speech or aphasia may result from disruption of any stage of this process, but for purposes of simplicity may be subdivided as follows:

- |                       |   |
|-----------------------|---|
| 1. Receptive aphasia  | { auditory (word deafness)<br>visual (word blindness or visual agnosia)           |
| 2. Expressive aphasia | { motor aphasia (loss of power of talking)<br>agraphia (loss of power of writing) |

Disorders of the use of words or syntax fall between these two groups, and it must be borne in mind that it is the exception to meet with a case of aphasia of a pure type. Thus, a patient may have both motor aphasia and also word-deafness; he may be unable to read as well as unable to write; and so on.

The *cortical centres* for the production and reception of speech are situated in the left cerebral hemisphere in right-handed persons, in the right hemisphere in the case of those who are left-handed. Hence the importance of ascertaining early in the investigation of a nervous case whether the patient is right- or left-handed.

The motor centre for spoken speech occupies the posterior extremity of the 3rd frontal convolution (Broca's convolution) and the lower end of the ascending frontal.

The centre for the production of written speech is believed to be in the posterior end of the 2nd frontal convolution.

The centre for the reception of spoken speech is in the posterior half of the superior temporo-sphenoidal convolution, and that for the reception of written speech (visual speech centre) extends from the posterior part of this convolution into the angular gyrus (see Fig. 47).

For practical purposes it is best to proceed with the investigation of aphasia in this order:

## 1 Spoken Speech

### How is it Received and Interpreted

First find out whether the patient's hearing is good. If so, ask him to put out his tongue, shut his eyes, etc. If he does so, test him as to his understanding of nouns by asking him to touch his nose, ear, chin, forehead, etc., in turn. Then test his verbs by asking him to smile, to whistle, etc. Finally, put to him longer questions, or give him more complicated orders, because when the disturbance of speech is only slight, he may be able to understand simple questions and commands, but not more complicated ones. If the patient responds satisfactorily to these tests, he has evidently no difficulty in interpreting the meaning of words heard—i.e. there is no *word-deafness*.

### How is it Produced?

i. If the patient can use only a *few words*, make a note of what these are. If he repeats any word or phrase again and again ('*recurring or repetitive utterance*'), note what it is.

ii. If he has a *considerable vocabulary*, first make a note of any examples of lalling, slurring, etc., as described at p. 220. This gives an indication of his *power of articulation*. Test him with such words and phrases as 'British Constitution', 'West Register Street', 'Biblical criticism', 'artillery'.

Secondly show him common objects—a knife, a pen, a matchbox, etc.—and ask him to name them: or, if he is dumb, to indicate with his fingers the number of syllables in the name of each. If unable to fulfil these tests, he has evidently got some forgetfulness of words (*nominal aphasia*). Sometimes the patient has a general idea of the word he wants to use, but forgets exactly how to pronounce it; he omits some syllables, or substitutes others for them, so that the listener may hardly be able to make out what word he wishes to use.

If he makes mistakes in his use of words, calling the knife a pen, or vice versa, he is suffering from *paraphasia*. In that case, one should note whether or not the patient shows that he is aware of his error by trying to correct himself, or whether he goes on talking gibberish.

### How is it Repeated or Echoed?

Ask him to repeat words after you. If he is word-deaf, try to make clear your request by the aid of pantomime, repeating the word or

phrase over and over again. If he is able to repeat what you say, endeavour to find out whether or not he understands what he is saying.

## 2 Written Speech

### How is it Received or Interpreted?

Ascertain whether or not his sight is good. If so, write on a piece of paper such questions or commands as, How old are you? Put out your tongue, etc. If he does not respond satisfactorily, there is some word-blindness present—i.e. the patient has *visual aphasia*. Inability to read is called *alexia*.

### How is it Produced?

Ask him to write his name. (This can often be done when all other power of writing is lost.) If he is able to do so, ask him some simple question—e.g. How many do two and two make?—and get him to write a reply. If he has word-deafness, put your question in writing. If his right hand is paralysed, make him write or print with his left. If he writes pretty well, get him to write an account of his illness, and note whether he makes use of the wrong word at times (*paraphasia*), or whether there is repeated use of any particular word.

### Can He Write to Dictation or Copy?

Try, using some simple book. If he succeeds, endeavour to ascertain whether or not he understands the meaning of what he writes.

## 3 Phenomena Associated with Speech

### Does He Understand Pantomime?

Does he nod his head for 'yes', shake it for 'no', and can he indicate numbers with his fingers? Loss of gesture language is termed *amimia*. Mistakes in the use of gestures—e.g. nodding for 'no', or shaking the head for 'yes'—are termed *paramimia*.

### Does He Understand Symbols?

One may write down certain numerals:

$$\begin{array}{r}
 2 & 2 & 2 \\
 2 & 2 & 2 \\
 \hline
 4 & 5 & 6
 \end{array}$$

and ask him to point out which is right. Inability to understand and manipulate mathematical symbols, termed *acalculia*, occurs in posterior parietal lesions affecting the dominant hemisphere. If he can read music, test him with musical notes.

### Can He Recognize Common Objects?

Place beside him a pencil, a coin and a match. Ask him to strike a light, or to write something down. If he is unable to select the proper article for the purpose, he is suffering from *mind-blindness* or *visual agnosia*, provided, of course, that his visual mechanism is intact. Inability to recognize his friends is another proof of the same condition.

It occasionally happens that a patient who has neither motor nor sensory paralysis, nor ataxia, cannot perform certain acts, though he can easily execute their component movements. He is consequently unable to make use of objects, though he can recognize their use. This condition is known as *apraxia*. It results from destruction of the left hemisphere, or of its connections, through the corpus callosum, with the right hemisphere. It affects only the left limbs, i.e. the right hemisphere, when the callosal fibres only are injured, but it is usually bilateral. It may be tested for by asking the patient to use certain objects, or make or imitate certain movements. For instance, he may be given a box of matches and a cigarette, and asked to light the latter. If there is apraxia, he may fail to open the box, or to take a match from it, or to strike the match, or even to light the cigarette with the match if he has succeeded in striking it. It is, of course, important to make sure that the patient understands the order.

## IV CRANIAL NERVE FUNCTIONS

In this section we propose to give a brief *résumé* of the essential points in the anatomy of each cranial nerve, to indicate its functions, and, in some cases, the chief symptoms which result from its paralysis, and then to describe the method in which one investigates the state of the nerve at the bedside.

### First or Olfactory Nerve

**Anatomy.** The central processes of the bipolar sensory cells in the olfactory epithelium pass through the cribriform plate to the olfactory bulb, where the cells of the second olfactory neurones lie. Nerve-fibres pass

thence to the olfactory area of the cerebral cortex, the uncus of the hippocampal gyrus.

**Test.** Have three small bottles containing some oil of cloves, some oil of peppermint, and some tincture of asafoetida. Apply these to each nostril separately, and ask the patient if he recognizes them. In testing, avoid the use of such irritating substances as ammonia, for these act partially through the 5th nerve. The sense of smell may be abolished. This is known as *anosmia*. Before concluding that the nerve is at fault, take care to exclude local changes in the nose itself—e.g. catarrh. *Parosmia* is the name applied to that condition in which the sense of smell is perverted, so that, for instance, offensive substances seem to have a pleasant odour, and *vice versa*.

Inquire also regarding *hallucinations of smell*. These sometimes constitute the aura of an epileptic fit.

### Second or Optic Nerve

**Anatomy.** From the retina, which is the end-organ of the sense of sight, the fibres of the optic nerve pass back to the optic chiasma. Here the fibres from the inner half of each retina decussate, whilst those from the outer half remain on the same side. Each optic tract, therefore, consists of fibres from the outer half of the retina on the same side and the inner half of the retina on the opposite side. Each tract passes back to the superior corpus quadrigeminum, the lateral geniculate body, and the pulvinar of the thalamus of the same side. In these most of the fibres of the optic tracts terminate. A further system of fibres, which is known as the optic radiation, takes origin in the lateral geniculate body and passes through the posterior limb of the internal capsule and then backwards to the cortex around the calcarine fissure. Those subserving the upper visual fields pass down through the substance of the temporal lobe, whilst those mediating impulses from the lower field pass up into the parietal lobe. The occipital cortex around the calcarine fissure constitutes the chief visual centre, and represents the opposite half of the field of vision, the left half of the field of vision being represented in the cortex of the right hemisphere, and *vice versa* (Plate XXIII).

**Test.** In testing the optic nerve, one has to investigate three functions: (1) Visual acuity; (2) visual fields; (3) colour sense.

Certain preliminaries must always be conducted. One of these is to see that any error of refraction in the patient's eye is first corrected and that also there is no gross disease of the structure of the eye; another is to take care to examine each eye separately.

### Visual Acuity

For technique of testing, see p. 286.

### Visual Fields

When we fix the eye upon an object, we not only see that object, but also a number of objects in the neighbourhood more or less distinctly. The full extent of this vision is measured in assessment of the visual fields. It should be noted, however, that the field of vision is limited both by the area of sensitive retina and by the margins of the orbit, nose and cheek. Hence the position of the eye is important. A rough estimate of the extent of the fields of vision for large objects may be obtained in the following way.

Seat yourself opposite to the patient and at a distance of about half a yard from him. If his right eye is to be tested, ask him to place his hand over his left, and to look steadily at your own *left* eye. Look steadily yourself at the patient's right eye, your own right being closed, and hold up your left hand in a plane midway between his face and your own, and at first at almost full arm's length to the side. Keep moving the fingers of the hand, and bring it nearer until you can just yourself 'with the tail of your eye' catch the movements of the fingers. Then ask the patient whether he sees them, telling him meanwhile to be sure not to take his own eye off yours. If he fails to see the fingers, keep bringing the hand nearer until he does see them. Test the field in this fashion in every direction—upwards, downwards to right, and to left—using the extent of your own field for purpose of comparison.

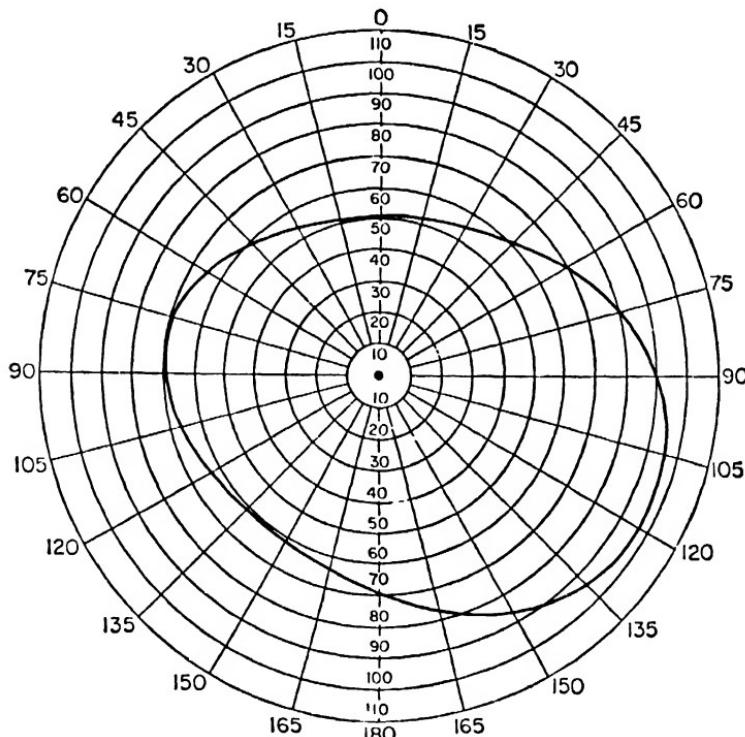
This gives the outline of his field for appreciation of a moving object, which may, however, be relatively intact when the fields for other forms of stimulation are seriously constricted. In consequence, his field for appreciation of a stationary object must also be investigated in a similar manner, but by asking the patient to indicate when he sees the observer's fingers held at rest.

Considering the field of vision in more detail, we appreciate that whereas the objects which cause images to fall upon the central part of the retina (the macula) are seen in minute detail and bright colouring, objects farther and farther from the point of fixation are seen with less and less distinction and colour, until at the periphery of the field we can only appreciate the presence of an object of considerable size without being able to judge its form. To the temporal side of the central point of vision is the *blind-spot* which represents the optic nerve head area in which there are no light receptors. Perimetry surveys the field of vision and the limits of perception are charted. The centre-point of the chart corresponds to the visual axis;

the point of fixation is therefore the point of more distinct vision. Around this point are arranged a series of more or less concentric lines, each of which denotes equal visual acuity, and is called an *isopter*. For purposes of investigation we divide the visual field into three parts:

- (1) An area surrounding the point of fixation to  $20^\circ$ .
- (2) An intermediate area between  $20^\circ$  and  $50^\circ$ .
- (3) An outer area from  $50^\circ$  to the periphery.

The fixation point is not exactly central, so that the outer and inner part of the field is unequally divided. Further, the boundary is delimited inwards and upwards by the nose and brow. Testing with



**Fig. 59. Extent of the right field of vision with a white target 20 mm. diameter mapped on a perimeter at a distance of 330 mm.**

## THE NERVOUS SYSTEM

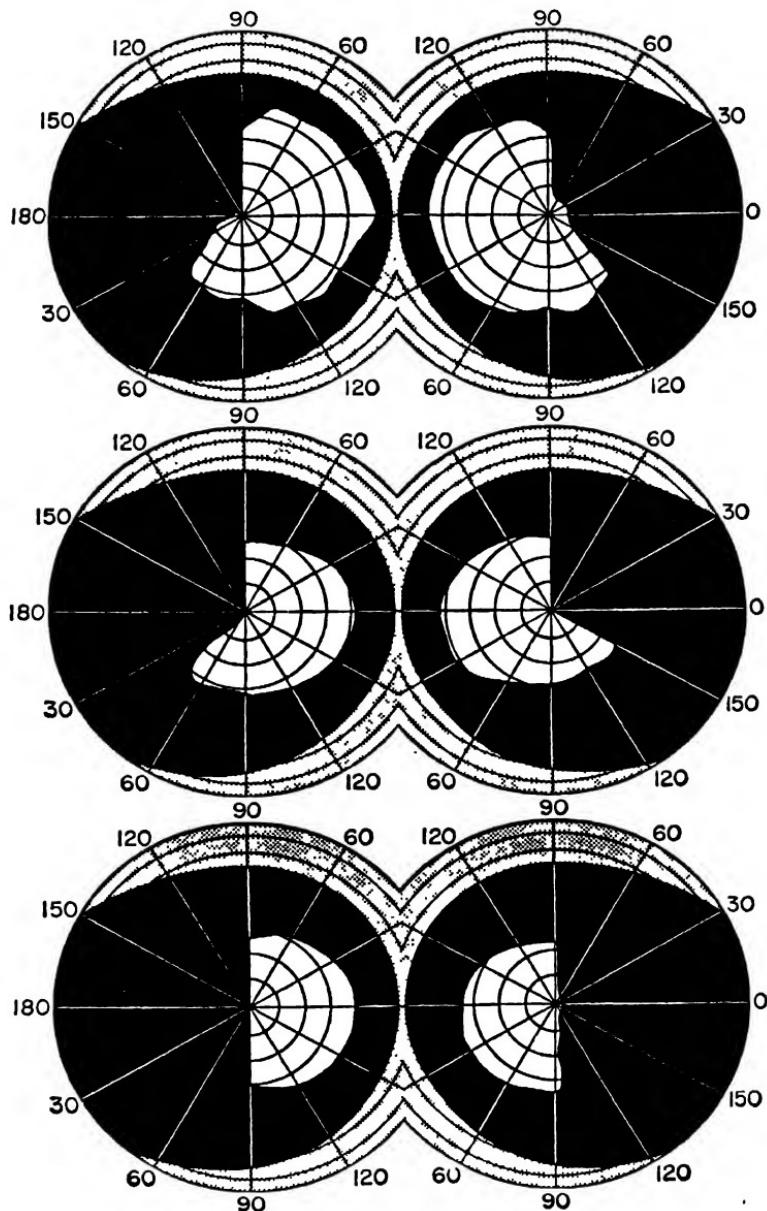


Fig. 61.

a 5 mm. object we find the extent of the average field of vision is 100° outwards, 60° upwards and inwards, and 75° downwards. The field charted with a 20-mm. object is shown in Fig. 59—note the restriction of the lower nasal field by the bridge of the nose.

The binocular field extends 200° or more laterally and about 140° vertically, in the middle of which is a circular portion common to each eye with a diameter of about 120°.

On each side of this paired area is a semilunar area which is unpaired and which accounts for the remainder of the field.

Perimetry is concerned with an investigation of the unioocular field of vision. We have noted that the visual acuity is very much lower at the periphery and gradually increases as we pass to the point of fixation. We may test this acuity with objects of different size, and we shall find that whereas a very small object is visible at and near the fixation point, it fades from view as it is withdrawn towards the periphery. By using a graduated series of objects, we are able to plot out a series of isopters each of which corresponds to a known size of object used at a known distance from the eye.

That part of the field between the periphery and the 30° circle is investigated by means of a perimeter:

A 3-mm. white test object is usually used, but when visual acuity is impaired, a larger diameter may be required. Thus the fraction of diameter of object/distance (usually  $\frac{3 \text{ mm.}}{330 \text{ mm.}}$ ) is a measure of the visual acuity at the particular point on the field which is being tested.

**Fig. 60. Fields of vision in a case of pituitary tumour (probably chromophobe adenoma), showing the development of a bitemporal hemianopia:**

- a. May 1947.  
VA Lt 6/12. Rt 6/18.
- b. August 1947.  
VA Lt 6/12. Rt 6/12.
- c. October 1947.  
VA Lt 6/12. Rt 6/12.

The fields were plotted on a Bjerrum's screen, at a distance of 2000 mm. from the eye to the point of fixation at the centre of the screen, using a 10-mm. white object. The outline of the black areas in the charts is that of the average normal field of vision and the white areas show the patient's fields.

(Courtesy of D. W. C. Northfield.)

The area within the  $30^{\circ}$  circle is examined by means of test objects upon a black screen—*Bjerrum's screen*—at a distance from the patient of 1000 or 2000 mm.

The patient is seated comfortably at this distance with the head steadied by a chin and head rest, and a grey object 1 cm. in diameter with a black centre is fixed to the screen on a level with the patient's eye.

The blind spot is first of all mapped out with a white object 20–30 mm. in diameter. The peripheral field is next mapped out with a 10-mm. object, and at a distance of 2000 mm. it should be circular and extend to about  $25^{\circ}$ , that is to the edge of the 2-metre square screen. With the small object areas of blindness or defective perception should be sought around the blind spot, especially between this area and the macula, the centrocaecal area, and in the horizontal meridian on the nasal side of the fixation spot. The findings are marked upon the screen with black pins, and subsequently transferred to a chart.

**Changes in the field of vision.** It may be contracted all round its periphery. This is spoken of as *concentric diminution* of the field of vision. It occurs in long-standing papilloedema, some forms of optic atrophy, bilateral lesions of the anterior part of the cortical visual centres, various affections of the retina, and in hysteria.

Sometimes the loss of vision is confined to the centre of the field. This is spoken of as a *central scotoma*. Sometimes it is due to local disease of the choroid or of the retina in the neighbourhood of the macula. In that case it may affect only one eye. A unilateral central scotoma is also produced by papillitis or retrobulbar neuritis, which in most cases is a symptom of disseminated sclerosis. It is sometimes due to toxic causes or vitamin deficiency, when it is generally bilateral. Pressure on the optic nerve is another cause. It may also result from a lesion of the posterior part of the cortical visual centres, and is then bilateral, but this is rare.

The term *hemianopia* means loss of sight in one-half of a visual field. When the same half of both fields of vision is lost, the hemianopia is described as homonymous, e.g. right homonymous hemianopia when the blindness occupies the right half of both the right and left fields (Plate XXIII).

*Superior* and *inferior hemianopia* means loss of the upper and lower halves of the visual field respectively. They are of rarer occurrence than the lateral variety, and are sometimes spoken of as altitudinal hemianopia. Hemianopia limited to one quadrant of the field is described as *quadrantic hemianopia* or *quadrantanopia*.

*Bitemporal hemianopia* means loss of vision in the temporal or

outer halves of both fields, and is due, therefore, to loss of visual power in the nasal half of each retina. It can only be produced by a lesion of the optic chiasma, involving those fibres of the optic nerves which decussate, and is accordingly rare. It is commonly due to a tumour of the pituitary body but may be produced by inflammatory or traumatic lesions of the optic chiasma (Fig. 60).

*Binasal hemianopia* signifies a loss of the nasal or inner half of each field, and indicates a diminution of visual power in the temporal half of each retina. It can be reproduced by a bilateral lesion confined to the uncrossed optic fibres on each side of the chiasma, and also may occur in open-angle glaucoma.

Temporal and nasal hemianopia are sometimes spoken of as *heteronymous*, in contradistinction to the *homonymous* variety.

### Colour Sense

For method of testing see Chapter 10, p. 287.

### Subjective Visual Sensations

Among the commonest subjective visual sensations is the occurrence of what are known as *muscae volitantes*—little specks seen floating before the eyes, especially on looking at a white surface or up to the sky. They most commonly occur in normal subjects, but are not infrequent in anaemic and debilitated persons. In migraine, peculiar zigzag lines, known as ‘fortification figures’, are often seen at the beginning of the attack, and in the investigation of such a case they should always be inquired for. The term *teichopsia* is applied to this condition. Hallucinations of sight occur in some cases, notably in delirium tremens; they may also form part of the aura in epilepsy.

### Third, Fourth, and Sixth Nerves

It is convenient to take these together, as conjointly they serve to innervate the muscles which move the eyeball.

**Anatomy.** The fibres of these nerves take their origin from a series of nuclei which begin in the floor of the aqueduct of Sylvius below the superior corpora quadrigemina, and extend down as far as the eminentia teres in the floor of the 4th ventricle. The nucleus for the 3rd nerve is farthest forward; its most anterior cells supply the ciliary muscle and iris, those for the ocular muscles being farther back. Behind that comes the nucleus of the 4th, and, most posteriorly of all, that of the 6th. The 3rd nerve emerges on the inner aspect of the crus, and is therefore apt to be involved in lesions implicating that part of the brain.

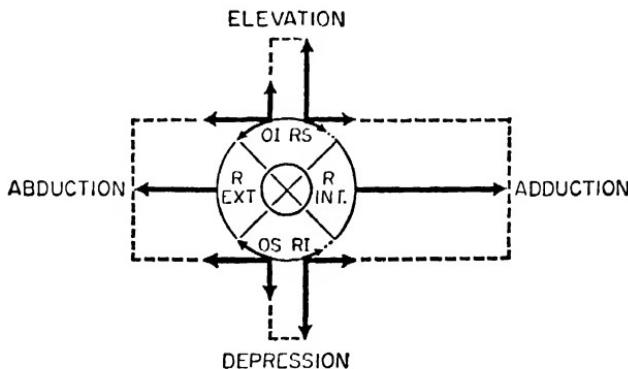
The 4th pair emerge on the anterior part of the roof of the 4th ventricle. They are peculiar in that they are the only cranial nerves which decussate between their nuclei and their point of emergence.

The 6th emerges between the medulla and pons. Its long intracranial course renders it particularly liable to the effects of pressure.

**Functions.** The 6th nerve supplies the external rectus, the 4th supplies the superior oblique. All the other extra-ocular muscles, along with the sphincter pupillae, the muscle of accommodation, and the levator palpebrae superioris, are supplied by the 3rd.

### Ocular Movements

Horizontal movement outwards is described as abduction, inwards as adduction; vertical movement upwards as elevation and downwards as depression. The eye is also capable of diagonal movements at any intermediate angle. Rotatory movements, the eye rolling like a wheel towards the nose (internal rotation) or away from the nose (external rotation), do not occur normally but may be seen in some varieties of ocular palsy. Fig. 61 shows the actions of the ocular



**Fig. 61. Scheme to illustrate the action of the ocular muscles.**

(R. Ext.—external rectus; R. Int.—internal rectus; OI—inferior oblique; RS—superior rectus; OS—superior oblique; RI—inferior rectus.)

*(After Marquex, from Fuchs.)*

muscles diagrammatically, the length of the arrows indicating the relative power of each muscle in different directions. It shows that the internal and external recti act directly in a single plane, but all movements require the co-ordinated activity of the whole group of extra-ocular muscles. Thus when the eye looks straight forward

elevation is the result primarily of the action of the superior rectus and superior oblique. In adduction the internal rectus is aided by the superior and inferior recti, and in abduction the external rectus is aided by the obliques. It will be seen that when two muscles act together in this way their conflicting actions cancel out, so that an orderly movement results.

Normally the movements of the two eyes are symmetrical, so that the visual axes meet at the point at which the eyes are looking. This is spoken of as conjugate movement of the eyes. Infra-nuclear (lower motor neurone) lesions of the 3rd, 4th and 6th nerves lead to paralysis of individual eye muscles or groups of muscles. Supra-nuclear lesions lead to paralysis of conjugate movements of the eyes.

### Symptoms and Signs of Paralysis

*Sixth nerve.* Inability to move the eye outwards and diplopia on looking in that direction. Possibly internal squint. In nuclear lesions there is also loss of the power of conjugate deviation of both eyes horizontally to the side of the lesion.

*Fourth nerve.* Impaired power of downward movement, and on the attempt to look downwards the eyeball is rotated inwards by the inferior rectus. Diplopia only below the horizontal plane, with the images uncrossed, but the false one tilted. There is rarely a visible squint.

*Third nerve.* Ptosis. The eye is displaced downwards and outwards, and further movement is only possible outwards and a little downwards. Pupil usually dilated and fixed. Loss of power of accommodation.

Paralysis of the 3rd nerve are not infrequently partial—only one or a few of these functions being lost.

In order to estimate degree of *ptosis*, one must eliminate the action of the occipito-frontalis. This is done by pushing down upon the latter muscle so that the eyebrows are kept level, and then asking the patient to look up. The extent to which the lids are raised indicates the strength of the levator. It must be remembered that smooth muscle in the upper lid is innervated by the cervical sympathetic and exerts a tonic elevating action. Slight ptosis therefore occurs after a lesion of the cervical sympathetic (*see p. 253*).

Any *retraction of the upper lid*, from over-action of the levator, should be noted by observing the relation of the edge of the lid to the upper margin of the cornea when the patient is looking straight forward.

Thus the signs of a lesion involving any of these nerves may be: 1, defective power of movement of the eye; 2, the presence of a squint; 3, the presence of diplopia. Of these signs the last is really the most trustworthy of all, for paralysis of the muscle(s) supplied by the nerve may be so slight as to lead to no appreciable squint and to no visible defect in mobility.

### Strabismus

By *squint* or *strabismus* is meant a condition in which the visual axes do not meet at the point of regard. Of this there are two varieties, *paralytic* and *concomitant*, and it is necessary that the two be carefully distinguished.

**Paralytic strabismus.** The following are the characters of a paralytic squint:

(1) *Limitation of movement.* Paralytic strabismus is due to loss of power in one or more of the extra-ocular muscles; a prominent feature, therefore, is lack of ability to move the eye in the direction of the physiological action of the muscle affected. Although this lack of power is usually apparent, sometimes the loss of muscular power is so slight, or the unaffected muscles mask the loss of action of the affected muscle so much, that the defective movement of the eye is hardly visible.

Movements in the so-called *cardinal directions* are tested by fixing the patient's chin with one hand and moving the forefinger of the other in the direction indicated. The eyes move normally 50° outwards, 50° inwards, 33° upwards and 50° downwards.

If an eye fails to move at all, or fails to move throughout the normal angular excursion, the deviation of the eye in a direction opposite to the physiological action of the muscle is called the *primary deviation* or *squint*, and it is measured by the angle which a line from the object to the nodal point of the eye makes with the visual axis. If now we cover the unaffected eye and so cause the patient to take on fixation with the affected eye, we shall find that the eye that is covered will deviate still more than the primary deviation of the affected eye. This deviation of the healthy eye is the so-called *secondary deviation* or *squint*, and this difference in amount between the primary and secondary deviation is the most important distinguishing feature between paralytic and concomitant strabismus.

(2) *False orientation of the field of vision.* This is an erroneous judgement by the patient of the position of an object in that portion of the field of vision towards which the paralysed muscle should normally move the eye. Take the case of a patient who has paralysis of the right external rectus muscle. If such a patient closes the left eye and is asked to touch suddenly an object held in the horizontal direction on his right side, he will fail and will strike wide of the object on his right-hand side.

The explanation of the phenomenon of secondary deviation being greater than primary is that when fixation is assumed by the paralysed eye, the same amount of nervous energy passes to the associated muscles of the two eyes, in this example, to the right external rectus and left internal rectus. As, however, the right external rectus is paralysed, an unusually great impulse is needed to stimulate it; the result is that its associated muscle in the left eye is over-stimulated and its movement too great.

This same reasoning may be applied to explain the phenomenon of false orientation, in that an object is projected into space largely according to the amount of nervous energy expended in moving the eye so as to fix that object. As in the case of paralysis the amount of nervous energy expended is in excess of movement, and so the object is projected too far in the direction of the physiological action of the paralysed muscle.

(3) *Vertigo* is occasionally a symptom of paralytic strabismus when both eyes are opened. It is due partly to the confusion of double sight and partly to false orientation.

(4) *Double vision (diplopia).* In order to overcome double vision, the patient turns his head in the direction of the action of the paralysed muscle. Information may therefore be obtained as to which muscle is involved by noting the way in which the head is held.

Patients with paralytic squint complain of double vision, which is due to the fact that owing to the lack of movement of one eye in a particular direction the images of external objects do not fall upon 'identical points' of the two retinae; this double vision or *diplopia* is therefore in that part of the field of vision into which the paralysed muscle should move the eye, were it unaffected.

In health, when fixing an object, the image formed in each eye falls upon the macula, so that not only are the two images of equal intensity and definition, but since they fall upon identical points of the two retinae they produce only a single image. In paralytic strabismus, the image of the object fixed falls upon the macula of the healthy eye and is seen with distinctness and detail, and is called the *true image*, whereas in the affected eye the image falls

upon the retina outside the macula, and as in consequence it is indistinct and blurred, it is called the *false image*.

**The investigation of a case of paralytic strabismus and the diagnostic value of diplopia.** First of all make certain that the diplopia is *binocular*, since certain conditions, lens opacities, astigmatism, etc., may produce *monocular* diplopia.

Movements in *cardinal* directions have already been mentioned, and they consist of movements up, down, in, and out. The main action of each individual muscle is in a cardinal direction, and so they are spoken of as lateral rotators, elevators, or depressors.

All the rectus muscles arise around the apex of the orbit and pass forwards to be inserted into the sclera, a varying distance behind the cornea. With the eyes in the primitive position, the external and internal recti turn the eyes to right and left only. Owing to the direction in which the superior and inferior recti pass to be inserted, although they act mainly as elevators and depressors of the eye, they also have a second component which causes them to act also as adductors.

The superior and inferior oblique muscles also act mainly as depressors and elevators respectively, but also act in a subsidiary way as abductors.

By turning the eye outwards 27° the superior and inferior recti may be made into almost pure elevators and depressors. Similarly by turning the eye inwards the superior and inferior oblique muscles may be made into almost pure elevators and depressors.

In this way we are able to resolve diplopia into horizontal and vertical (which much simplifies our investigation), and find the field of maximum diplopia in one of the cardinal directions.

When the images in diplopia are separated laterally, so that the right image belongs to the right eye and the left to the left eye, the condition is spoken of as *homonymous* or *uncrossed diplopia*. If, however, the left image belongs to the right eye and the right to the left, it is called *heteronymous* or *crossed diplopia*. It will also be found that the real image belongs to the healthy eye, whereas the false image belongs to the paralysed eye.

**The production of homonymous diplopia.** If, as the result of paralysis of an abductor muscle (external rectus muscle), there is deviation of the eye inwards (convergent strabismus), the image in this eye will fall upon a point of the retina internal to the macula. Two things will result: (1) the image will not be so sharp as the image in the healthy eye, proving that the *false image* belongs to the affected eye; (2) since images that fall upon the retina on the nasal side of the

macula are projected in space to the temporal side of the eye, it follows that *paralysis of an abductor producing convergent strabismus causes homonymous diplopia* and also that the false image is projected in the direction of the physiological action of the paralysed muscle.

**The production of heteronymous or crossed diplopia.** If, as the result of paralysis of an adductor muscle (internal rectus), there is a deviation of the eye outwards (divergent strabismus), the image in this eye will fall upon a point of the retina external to the macula. The false image is produced in the affected eye, and since images that fall upon the retina to the temporal side of the macula are projected into space to the nasal side of the eye, it follows that *paralysis of an adductor producing divergent strabismus causes heteronymous or crossed diplopia*.

In a similar way it may be shown that in paralysis of an elevator muscle the false image (which belongs to the affected eye) lies on a higher level than the true image, and in paralysis of a depressor muscle the false image lies below the true image.

If a rotator muscle is weak, the false image is tilted.

**Actions of the muscles.** The external and internal recti muscles move the eyes in a horizontal direction, consequently maximum diplopia is produced when the eyes turn horizontally in the direction towards which the paralysed muscle normally turns the eye.

We have seen that the superior and inferior recti muscles become simple elevators and depressors when the eyes are turned outwards, consequently maximum vertical diplopia is produced when the eyes look up and out and down and out respectively.

The inferior and superior oblique muscles become simple elevators and depressors when the eyes are turned strongly inwards, consequently maximum vertical diplopia is produced when the eyes look up and in and down and in respectively.

In conjugate movements the muscles of the two eyes act in pairs (for instance, in conjugate movements in the horizontal direction to the right, the right external rectus muscle is linked in action with the left internal rectus muscle) and the table below shows a series of six pairs, each pair representing 'true associates':

**1. Muscles moving the eyes laterally.**

(a) To the right:

Right external rectus,  
Left internal rectus.

## (b) To the left:

Left external rectus.  
Right internal rectus.

2. *Muscles moving the eyes upwards.*

## (a) With the eyes turned to the right:

Right superior rectus.  
Left inferior oblique.

## (b) With the eyes turned to the left:

Left superior rectus,  
Right inferior oblique.

3. *Muscles moving the eyes downwards.*

## (a) With the eyes turned to the right:

Right inferior rectus,  
Left superior oblique.

## (b) With the eyes turned to the left:

Left inferior rectus,  
Right superior oblique.

4. *Muscles which rotate the eyes.*

## (a) External rotators:

Inferior rectus,  
Inferior oblique.

## (b) Internal rotators:

Superior rectus.  
Superior oblique.

**Method of finding the direction of maximum diplopia.** The patient is seated with the head fixed in position (preferably in a head-rest) with a red glass before the right eye, and a green glass before the left. At a distance of about 15 ft. the observer moves a light in the direction indicated in the diagram on p. 239, each of the lateral squares corresponding to a pair of true associated muscles; thus a maximum vertical diplopia produced when the patient looks up and to the right into the right superior square shows that either the right superior rectus or the left inferior oblique muscle is the one affected. It is only necessary to find to which eye the false image belongs to decide which muscle is paralysed. The higher of the two images indicates the affected eye.

Note that in using this chart the area of greatest vertical diplopia is the namesake either of the affected muscle or its true associate in

the other eye; further, that the true associates always bear names which are the most contrary possible, thus *left inferior oblique* is in every term opposite to *right superior rectus*.

L	The patient looks	R
Upwards to the left LEFT SUPRIOR AREA	Upwards SUPERIOR MEDIAN AREA	Upwards to the right RIGHT SUPERIOR AREA
To the left LEFT EXTERNAL AREA	Straight ahead PRIMARY AREA	To the right RIGHT EXTERNAL AREA
Downwards to the left LEFT INFERIOR AREA	Downwards INFERIOR MEDIAN AREA	Downwards to the right RIGHT INFERIOR AREA

Table for charting the field of diplopia in a case of paralytic strabismus.

### Concomitant Strabismus

It has been explained when dealing with paralytic squint that the amount of angular deviation of the two visual axes varies with different positions of the two eyes, and also that secondary deviation is always greater than primary deviation.

In concomitant strabismus, as its name implies, the angular deviation of the visual axes is the same in whatever position the eyes may be; in other words, the primary and secondary deviation are always equal.

*Cover test.* Ask the patient to look at an object immediately in front of him. Suddenly cover the apparently fixing eye and ask the patient to fix the object with the uncovered eye. If this eye makes any movement in taking up fixation, it must have been previously deviating. If now the eye behind the screen (which was previously

fixing) is observed it will be seen to deviate in the same relative direction as was the other eye, and *to the same angular amount*, that is, the primary and secondary deviation are equal.

**The Clinical Features of Concomitant Strabismus are:**

- (1) It always begins in early childhood, over 70 per cent before the 5th year and the great majority before three years of age.
- (2) The movements of the eyes are good in all directions.
- (3) Diplopia is practically never a symptom.
- (4) The primary and secondary deviation are equal.
- (5) The deviating eye often has defective vision.

A squint may be *intermittent* or *constant*, and if constant, *monocular* when the same eye deviates whilst the other usually fixes, or *alternating* when either eye fixes indifferently.

**Nystagmus.** The term nystagmus is applied to a disturbance of ocular posture characterized by involuntary, rhythmical oscillations of the eyes. These movements may be horizontal, vertical, or rotary. The speed of the movements may be the same in both directions, or quicker in one direction than another; in the latter case the quicker movement indicates the direction of the nystagmus. To examine for nystagmus, ask the patient to look straight in front of him, and observe whether the eyes remain steady. Then ask him to look to his extreme right, then to the left, and then upwards and downwards. Observe the rate, amplitude and rhythm of the nystagmoid movements in each direction, and whether they are influenced by the position of the head (positional nystagmus).

A few irregular jerks of the eyes are often seen in full lateral deviation. The brief duration and irregularity of these movements distinguish them from true nystagmus.

Nystagmus is most commonly due to visual disorders, affections of the vestibular system (either centrally or peripherally), lesions affecting the central pathways concerned in ocular posture, or weakness of the ocular muscles. Nystagmus of visual origin is pendular and often rotary on central fixation of the eyes. Congenital nystagmus sometimes occurs without discernible cause and it also shows this pendular quality.

**Conjugate ocular palsies.** In addition to the defects of movement due to paralysis of the individual ocular muscles, weakness or paralysis

of the movement of both eyes in one direction sometimes occurs. Thus the patient may be unable to look to either side, or upwards or downwards; or the power of convergence alone may be lost. Weakness of conjugate lateral movement may occur in hemiplegia due to cerebral lesions, especially in the acute stage. Palsy of this movement occurs with lesions in the neighbourhood of the 6th nucleus of the side to which the movement is weak. Bilateral paralysis of lateral conjugate movement is seen in centrally placed pontine lesions above the level of the 6th nerve nuclei. The conjugate vertical palsies are always associated with disease of the corpora quadrigemina, or in the neighbourhood of the oculomotor nuclei.

If both eyes are kept persistently turned in one direction, the condition is spoken of as *conjugate deviation* of the eyes. It is usually either to the right or to the left. Conjugate deviation of the eyes may be brought about either by a lesion which produces paralysis or by one which causes irritation or spasm. In the former case the eyes (and usually also the head) are turned towards the side of the lesion, provided the latter is in the cerebral hemisphere. The patient, in fact, is said 'to look towards his lesion'. An irritative lesion in a similar situation causes the deviation to be towards the healthy side. If, however, the lesion has its seat in the pons, these rules are reversed, the deviation being towards the sound side in a paralytic lesion, and towards the affected side in one which is irritative.

**Skew deviation of the eyes**—in which, for example, one is directed upwards and the other downwards—occurs in certain lesions of the labyrinth, 8th nerve and cerebellum.

#### Examination of the Pupils

The following points must be noted about the pupils in every case:

##### 1 Size

Compare the size of the two pupils, first in a bright light and then in a dim light. Note whether the pupils are large or small, and whether any irregularity is present. It must be remembered that the size of the pupil in health is subject to great variation. As a rule, the pupils are larger in dark eyes than in light. They tend to be small in elderly subjects. Slight inequality of the pupils may also be present in perfectly healthy subjects.

If one pupil is larger than the other, one must decide which is the

normal. This is not always very easily answered, but, as a rule, the pupil which exhibits the less mobility is to be regarded as the abnormal one.

## 2 Shape

Note whether the pupil is circular in outline, as it should be, or whether its contour is irregular. Such irregularities may be due to adhesion of the iris to the lens, as a result of an old iritis. Irregularity in shape of the pupil is often an early sign of neurosyphilis, and of third-cranial-nerve palsy.

## 3 Mobility

(a) **Reaction to light.** This is a reflex action. The afferent fibres involved are contained in the optic nerve, travelling to the oculomotor nuclei whence the efferent fibres pass by the 3rd nerve, through the ciliary ganglion, to the pupil-sphincter.

**Test.** Examine each eye separately. Place the patient opposite a bright light, be sure his accommodation is relaxed, and cover the eye with the hand. Leave it covered until the pupil dilates, then withdraw the hand and watch the pupil closely. It should contract almost immediately, then dilate again a little, and, after undergoing slight oscillations, settle down to its normal size. The test may also be carried out by concentrating light upon the pupil with an electric torch.

Owing to the decussation of some of the fibres of the optic nerves at the chiasma, light acting upon one eye affects the centre for pupil contraction of the other eye as well as that of its own side. Fibres concerned in the reflex also decussate in the mid-brain. As a consequence, if light is shut off from one eye both pupils dilate, and if bright light is made to enter one eye both pupils contract. This is known as the *consensual reaction* of the pupils. It should be tested by keeping one eye in the shade while light is thrown into the other. The effect on the pupil of the shaded eye is then observed.

(b) **Reaction to accommodation.** The pupils become smaller on accommodating for a near object. Convergence of the eyes, accommodation and contraction of the pupils are due to associated muscular contractions. The contraction of the pupils is thus also described as the reaction on convergence and the reaction on accommodation-convergence.

*Test.* Hold up one finger close to the patient's nose. Ask him to look away at a distant object. Then suddenly tell him to look at your finger. As the eyes converge to accomplish this the pupils should become decidedly smaller.

If the patient is unable to see, the test may still be carried out by getting him to hold up his own finger about a foot in front of his face, and then asking him to direct his eyes to it.

*Argyll-Robertson pupil.* This is the term applied to the condition of pupil usually observed in neurosyphilis. It reacts to accommodation, but not to light. Sometimes the reaction to light is not entirely absent, but takes place in a very sluggish fashion. The Argyll-Robertson pupil is also small and irregular, and dilates slowly and imperfectly to mydriatics. *Hippus* is the term applied to the alternate contraction and dilatation of the pupil, which can sometimes be observed going on rhythmically. It is of no known significance.

(c) **The myotonic pupil.** This is a pupillary abnormality usually associated with absent tendon reflexes in the legs. The affected pupil is generally larger than normal. The reaction to light is lost, or almost completely so, and that to accommodation is very slow.

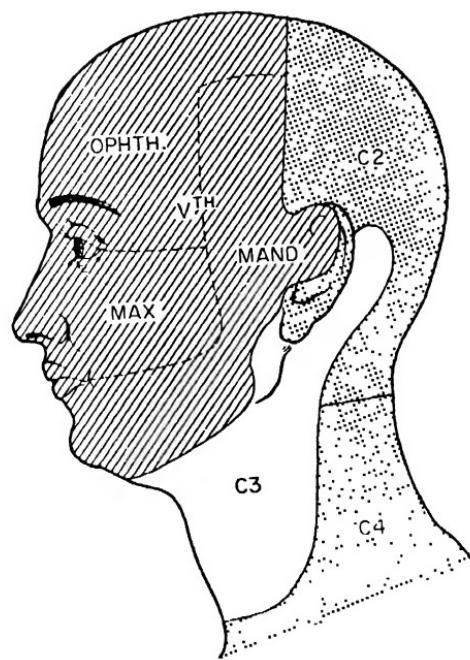
(d) **Cilio-spinal reflex.** Dilatation of the pupil can often be observed to follow irritation of the skin of the neck either by pinching or by the action of a faradic current. It is due to reflex excitation of the pupil-dilating fibres in the cervical sympathetic (p. 253), and is abolished in lesions of that nerve.

### Fifth Nerve

The **sensory root** takes origin from the cells of the Gasserian ganglion and enters the lateral surface of the pons at about its middle. The fibres which conduct impulses for light touch and postural sensibility terminate in a large nucleus in the pons situated near the floor of the 4th ventricle and lying externally to the motor nucleus, while the fibres for pain and thermal sensibility terminate in the 'descending' or bulbospinal root, which extends as low down as the 2nd cervical segment of the cord. Immediately beyond the Gasserian ganglion the nerve separates into its three divisions.

The *first or ophthalmic division* supplies the conjunctiva (except that of the lower lid), the lachrymal gland, the mesial part of the skin of the nose as far as the tip, the upper eyelids, the forehead, and the scalp as far as the vertex.

Paralysis of this division results in loss of sensibility in the area of skin and mucous membrane supplied, and may cause trophic



**Fig. 62.** Lateral view of the skin areas supplied by the Vth cranial nerve and the 2nd, 3rd and 4th cervical segments.

changes in the eyeball, *neuropathic keratitis*. The corneal reflex is abolished.

The *second or maxillary division* supplies the cheek, the front of the temple, the lower eyelid and its conjunctiva, the side of the nose, the upper lip, the upper teeth, the lining membrane of the nose, the upper part of the pharynx, the roof of the mouth, most of the soft palate, and the tonsils.

Paralysis of this division leads to abolition of sensibility in the above area, and loss of the palate reflex.

The *third or mandibular division* is joined by the motor root. It supplies sensation to the lower part of the face, the lower lip, the ear, the tongue, and the lower teeth. It supplies also the salivary glands and, through the motor division, the muscles of mastication, the tensor tympani, and also, perhaps, the tensor palati, although many believe that this muscle is innervated by the spinal accessory.

**2. Motor root.** This takes origin in a small nucleus lying internally to the chief sensory nucleus, and partly also from the mesencephalic root, which arises in nerve-cells scattered around the aqueduct of Sylvius. It emerges at the side of the pons, just in front of the sensory division, passes underneath the Gasserian ganglion, and joins the inferior maxillary division, to which it gives its motor fibres.

Paralysis of the whole 5th nerve leads to loss of sensation in the areas of skin and mucous membrane above mentioned, and to defective power of chewing (Fig. 62). Trophic lesions may be present, and the salivary, buccal and lachrymal secretions much diminished; the sense of taste is occasionally abolished on the interior two-thirds of the same side of the tongue.

One curious result of the sensory paralysis is that the patient, when drinking, imagines that the cup is broken, as he only feels it on one side of his mouth.

### How to Test the Fifth Nerve

**1 Motor functions.** Ask the patient to clench his teeth while the observer keeps his hands on the temporal and then on the masseter muscles. These should stand out with equal prominence on each side. If there is paralysis on one side, the muscles on that side will fail to become prominent. On opening the mouth the jaw deviates towards the paralysed side, being pushed over by the healthy external pterygoid muscles. The condition of the tensor tympani muscle cannot be satisfactorily examined except by noting whether there is any difficulty in hearing notes of a particular pitch.

**Sensory functions.** The common sensibility of the area supplied is tested in the usual way (p. 265).

**Taste.** In suspected lesions of the 5th nerve the sense of taste should always be examined, as it seems probable that, in certain cases at least, taste fibres from the anterior two-thirds of the tongue reach the brain through the 5th nerve. As a rule, however, they pass from the lingual nerve into the chorda tympani, and thence through the geniculate ganglion and the nervus intermedius of Wrisberg into the medulla oblongata. The taste fibres from the posterior third of the tongue enter by the glossopharyngeal nerve.

All the taste fibres enter the tractus solitarius and relay in the nucleus of the tract, whence further fibres pass upward in the gustatory fillets to the thalamus and thence to the foot of the post-central gyrus. *Ageusia* or loss of taste occurs with lesions of the peripheral pathways concerned or with centrally placed pontine lesions which may involve the gustatory fillets.

*How to test the sense of taste.* Have some sugar, some quinine, and some salt, all in powder. Ask the patient to put out his tongue and to keep it out until the conclusion of the test. Many men, especially smokers, are unable to taste on the protruded tongue. The tongue should then be drawn in, but the mouth kept open in order to avoid spread of the test substances. Dry the tongue, place some sugar on it, rub it gently in, and ask, 'Is that salt?' If taste is normal he will shake his head. In this way all the substances are tried, first on the anterior part of the tongue and then at the back. A weak galvanic current is also a useful test. It should produce a sort of metallic taste.

Alternatively, use strong solutions of sugar and common salt, and weak solutions of citric acid and quinine. These are applied by a glass rod to the surface of the protruded tongue, and if the taste is recognized the patient writes down 'sweet', 'salt', 'sour', or 'bitter', as the case may be, without withdrawing the tongue. After each test the mouth must be rinsed. The quinine test should be applied last, as its effect is more permanent than that of the others.

Loss of taste may, of course, arise from lesions of the taste fibres in any part of their course.

In addition to loss of taste, one should always ask the patient whether he has any abnormal taste sensations. These may form the aura of an epileptic fit.

**Reflex functions.** The corneal reflex (*see p. 272*).

### Seventh Nerve

**Anatomy.** The course of the fibres from the cortical centre to the nucleus of this nerve has already been described (p. 199). The nucleus is situated in the pons lateral to that of the 6th nerve. On leaving the nucleus the fibres wind round the nucleus of the 6th, and finally emerge medially to the 8th nerve, between the olive and restiform bodies.

The nerve lies in close contact with the 8th, so that a lesion of the one at this part can hardly avoid injuring the other, and enters the internal auditory meatus along with it. During its course through the temporal lobe, in close proximity to the aditus of the tympanic antrum, it gives off a branch to the stapedius muscle. It is joined by the chorda tympani at the geniculate ganglion, which contains taste fibres from the anterior two-thirds of the tongue. In this part of its course the nerve is exposed to the effects of pressure, owing to its being enclosed in a bony tube. It emerges at a point opposite the junction of the anterior border of the mastoid with the ear, and spreads out on the side of the face to supply its muscles.

**Functions.** The 7th is a purely motor nerve. It supplies all the muscles of the face and scalp, except the levator palpebrae superioris. It also supplies the platysma.

**Effects of paralysis.** These are usually at once seen on looking at the patient. The affected side of the face has lost its expression. The naso-labial fold is less pronounced, the furrows of the brow are smoothed out, the eye is more widely open than the other, and the mouth is somewhat drawn to the healthy side. The patient is unable to whistle; food is apt to collect between his teeth and his gums, and saliva and any fluid he drinks may escape from the affected angle of the mouth.

### How to Test the Seventh Nerve

1. Ask the patient to shut his eyes as tightly as he can. Note that the affected eye is either not closed at all—in which case the eyeball rolls upwards to make up for the failure of the lid to descend—or, if the eye is closed, the eyelashes are not so far rolled in as on the healthy side. Try also forcibly to open the eyes while the patient attempts to keep them closed. If the orbicularis is acting normally, it should be almost impossible to open the eye against the patient's wish. If the muscle is partially paralysed, however, the exertion of very little force may open it.

The effect of screwing the eyes tightly shut causes the corners of the mouth to be drawn upwards. In paralysis of the lower part of the face, the corner on the affected side is either not drawn up at all, or not so much as on the healthy side.

2. Ask the patient to whistle. He is unable to do so.
3. Ask him to smile or show his upper teeth. The mouth is then drawn to the healthy side.
4. Ask him to inflate his mouth with air and blow out his cheeks. Tap with the finger in turn on each inflated cheek. Air can be made to escape from the mouth more easily on the weak or paralysed side.

### Signs of Paralysis of the Facial Nerve in Different Parts of its Course

Paralysis of the face presents different symptoms depending on whether the lesion is situated above the nucleus, at the nucleus or below it. The former constitutes what is known as upper motor neurone or supranuclear facial paralysis, the latter produces lower motor neurone or infranuclear paralysis.

The chief difference between the two forms is that in *supranuclear paralysis* the lower part of the face is chiefly affected; in *infranuclear*

*paralysis* both the upper and lower parts are equally involved. The explanation of this is that there is bilateral innervation of the muscles of the upper part of the face, and consequently a unilateral lesion only partially cuts off the nerve impulses to one side. Sometimes a supranuclear lesion only affects the fibres concerned in emotional movement and this function should be tested separately from voluntary movement.

**Infranuclear facial paralysis** may be produced by a lesion of the nucleus or of the facial nerve itself.

A lesion inside the facial canal—unless it is towards the outer end—involves the fibres of the chorda tympani, and therefore produces loss of taste sensation in the anterior two-thirds of the tongue.

Lesions of the nucleus or the nerve below it will result in atrophy of the facial muscles. Supranuclear lesions do not produce this effect. Bilateral weakness of the face is sometimes difficult to detect, especially if it is quite symmetrical.

**Abnormal facial movements.** The muscles supplied by the facial nerve are frequently affected by spasm or spasmodic movements. These may involve all the facial muscles, or groups of them only. The spasm may be of either the clonic or tonic variety (p. 261). If present, the nature of the movements, their extent, and the muscles affected by them should be carefully noted.

### Eighth Nerve (Auditory)

**Anatomy.** This nerve consists of two sets of fibres. One set supplies the cochlea, and subserves the function of hearing; the other part supplies the vestibule and semicircular canals, and is the nerve of equilibration. The *auditory* fibres, which arise from the cochlear ganglion, enter the brain-stem at the lower border of the pons and are distributed to the dorsal and ventral cochlear nuclei. The *vestibular* fibres take origin from the vestibular ganglion, and terminate in a group of nuclei in the pons and medulla.

The secondary auditory tracts, after partial decussation, terminate in the inferior corpora quadrigemina and the medial geniculate bodies, and another system that takes origin from these passes through the internal capsule to the cortical centre for hearing, in the 1st and 2nd temporo-sphenoidal convolutions. Sounds received in one ear probably reach the opposite hemisphere of the brain predominantly, but owing to the partial decussation of the secondary auditory tracts neither unilateral cerebral nor brain-stem lesions produce deafness in one ear.

The vestibular nerve is closely connected with the cerebellum. It has cerebral connections but these are probably not very important.

**Tests of Hearing.** Before testing a patient's power of hearing, exclude the presence of wax in the ear (*see p. 305*). This done, the hearing-power can be tested by means of a watch. Stand behind the patient and ask him to shut his eyes. Begin outside the probable range of hearing power, and bring the watch gradually nearer the ear, asking the patient to speak when he hears the tick. It is necessary to know at what distance the tick should be audible to a healthy ear. Test each ear separately, keeping one closed whilst the other is being examined.

If impairment of hearing is detected, it must be determined whether it is really due to disease of the auditory nerve or merely to some affection of the middle ear. In order to settle this point, the *tuning-fork* test may be employed. When the fork is beating strongly, hold it opposite the ear; if it can be heard, then place its base on the mastoid process in order to determine if its vibrations can be heard when conducted through bone. If the patient hears this, ask him to compare the relative loudness of the fork when heard through air and through bone, or to determine which can be heard the longer as the vibrations die out. This is *Rinne's test*. Normally, aerially conducted sounds are louder to the patient than those conducted through bone. In middle-ear disease, aerial conduction is diminished or lost, while bone conduction remains more or less normal. When the auditory nerve is affected, both air and bone conduction are diminished or lost.

*Weber's test*, though less reliable than *Rinne's*, should also be used. Strike a tuning-fork and place the end of it against the centre of the patient's forehead. If the deafness discovered by the watch is due to an affection of the middle ear, the patient will hear the tuning-fork better on that side than on the healthy one. On the other hand, if the deafness is due to disease of the auditory nerve, the tuning-fork will only be heard on the healthy side. The test may also be carried out by means of the watch. In affections of the nerve, the watch is not heard even when pressed against the ear; in disease of the middle ear, it is heard even more loudly than when similarly applied to the healthy side. The explanation of these facts is not yet clear, nor are the tests invariably trustworthy. They hold good, however, for most cases. Other points in favour of the deafness being due to a lesion of the nerve and not to disease of the middle ear are: (a) if the hearing

is better in a quiet place, (b) if conversation is heard better than the watch, (c) if inflation of the middle ear renders the hearing worse.

**Abnormal auditory sensations.** The patient may complain of 'ringing in the ears', or *tinnitus*. The precise character of the sound varies in different cases. It may be of a humming, buzzing, hammering or whistling character. The presence or absence of this symptom should always be inquired for, and whether it is constantly present, or in what circumstances it occurs.

Hyperaesthesia of the auditory nerve (*hyperacusis*), by which even slight sounds are heard with painful intensity, sometimes occurs, especially in hysteria and in lesions of the facial nerve above or in the aqueduct, owing to paralysis of the stapedius muscle.

*Hallucinations of sound* may also be present, the patient fancying that he hears voices, bells, etc. These occur chiefly in states of mental disturbance, but they sometimes arise as the aura of an epileptic attack when the causal lesion is situated in or near the auditory cortex.

**Tests for Vertigo.** The patient will describe this as giddiness or dizziness. In order to constitute true vertigo, external objects should seem to move round him. Ask if this is the case, and if so, in what direction the objects seem to move. Ask also whether the vertigo causes him to fall to the ground.

Vertigo is usually due to an affection of the vestibular system, but weakness of an ocular muscle occasionally gives rise to it. When a patient complains of vertigo one should look for signs of disease of the ear, 8th nerve, or brain-stem.

#### Ninth (Glosso-Pharyngeal), Tenth (Vagus) and Eleventh (Spinal Accessory) Nerves

**Anatomy.** These arise in order from above downwards from an elongated nucleus in the floor of the 4th ventricle. They emerge by several roots along the lateral aspect of the medulla, beginning above in the groove between the olive and restiform bodies. The spinal part of the 11th emerges from the lateral column of the cord, beginning as low as the 6th cervical nerve; it passes up through the foramen magnum to join the medullary (accessory) part, and emerges with it through the jugular foramen. After its emergence the two divisions of it again part company, the medullary or accessory portion joining the vagus, supplying motor fibres for the larynx and pharynx.

The *eleventh* nerve is purely motor in function, contributing to the innervation of the larynx and pharynx as well as the sternomastoid and

trapezius. The spinal part of the nerve dips beneath the sternomastoid muscle about one inch below the tip of the mastoid process, and emerges from underneath that muscle again at about the middle of its posterior border. It supplies the sternomastoid and trapezius, which are also supplied by twigs from the cervical plexus.

The *ninth* (glosso-pharyngeal) is sensory for the posterior third of the tongue and for the mucous membrane of the pharynx. It is motor for the middle-constrictor of the pharynx and for the stylopharyngeus. It contains the taste fibres for the posterior part of the tongue (see p. 245).

**How to test the glossopharyngeal.** The 9th nerve is rarely paralysed alone. Paralysis can best be diagnosed by examining its sensory and reflex functions. Examine the power of taste in the posterior part of the tongue (p. 245). Loss of it *may* mean paralysis of the trunk of the glossopharyngeal nerve.

Tickle the back of the pharynx, and note if reflex contraction occurs.

The *tenth* (vagus) is motor for the soft palate (with the exception of the tensor palati), pharynx, and larynx. It is also sensory and motor for the respiratory passages, the heart, and (through the sympathetic ganglia) for most of the abdominal viscera.

The fibres for the soft palate, pharynx, and larynx take origin in the nucleus ambiguus, emerge in the upper roots of the 11th, reach the pharyngeal plexus, and thence pass to the muscles of the palate, the constrictors of the pharynx, and to the larynx.

The viscero-motor and the cardio-inhibitory fibres are derived from the dorsal vagus nucleus in the floor of the 4th ventricle.

**How to test the vagus.** Paralysis of the vagus is chiefly made evident through its palatine and laryngeal branches.

1. *The palate.* Ask the patient whether he is troubled with the regurgitation of fluids through his nose when he tries to swallow. This is a common occurrence in total paralysis of the soft palate, owing to defective elevation during swallowing. For a similar reason the patient is unable to pronounce words which require complete closure of the naso-pharynx. Thus 'egg' is sounded as 'eng', 'rub' becomes 'rum', and so on. In unilateral paralysis these symptoms are not observed.

For direct examination of the soft palate, place the patient facing

the light with his mouth open, and introduce a tongue depressor. The position of the uvula is quite unreliable as a guide to the state of the soft palate, as deviation of it is not uncommon even in health. One must watch the movements of the palate during phonation. Ask the patient to say *Ah!* and observe whether both sides of the palate arch upwards; in health, elevation of the palate will occur when the patient says *Ah!* If one side is paralysed, that side will remain flat and immobile, and the median raphe will be pulled towards the other side. The manner in which the palate rises in such a case has been compared to the ascent of a curtain of which one string is broken. In bilateral paralysis the whole palate remains motionless.

**2. The larynx.** The superior laryngeal branch of the vagus is sensory for the larynx above the level of the true cords, and is motor for the crico-thyroid muscle. Unilateral paralysis of the nerve does not produce any symptoms. Bilateral paralysis causes the vocal cords to be relaxed. The voice is therefore hoarse and deep, and the utterance of high notes impossible.

The recurrent laryngeal branch supplies sensation to the larynx below the level of the cords, and motor fibres to all the laryngeal muscles except the crico-thyroid. Paralysis of it leads to appearances which are recognized by the laryngoscope, and are described on p. 309.

**How to test the spinal accessory.** Paralysis of the upper part of the trapezius is evinced by asking the patient to shrug his shoulders while the observer offers passive resistance by pressing on the shoulders from behind. Paralysis of the sternomastoid causes weakness in rotation of the chin towards the opposite side.

#### Twelfth or Hypoglossal Nerve

**Anatomy.** The 12th nerve arises from a nucleus in the lower part of the floor of the 4th ventricle, close to the middle line. It emerges between the anterior pyramid and the olive. It is a purely motor nerve, supplying the tongue and the depressors of the hyoid bone.

**Test.** Ask the patient to put out his tongue as far as possible. If the hypoglossal is paralysed, the tongue, instead of being protruded straight, is pushed over to the paralysed side. Be careful not to mistake an apparent deviation of the tongue, really due to the mouth being twisted to one side, for a real deviation. Such an apparent

deviation occurs in facial paralysis. Ask him also to move his tongue from side to side, and to lick each cheek with it; observe whether he can do so freely. Note whether there is any wasting of the tongue, and whether there is any tremor or fasciculation in it. The presence of wasting indicates that the lesion is either nuclear or infranuclear.

*Paralysis of the cervical sympathetic* may be considered here. A complete description of the functions and distribution of the nerve, however, is not necessary in such a work as this. For purposes of diagnosis the fibres supplied to the eyeball are alone of importance. These take origin in the lower cervical and upper thoracic regions of the spinal cord (cilio-spinal centre), from which the fibres emerge in the first thoracic nerve-roots and pass to the sympathetic cord by the rami communicantes. From the cervical sympathetic cord the fibres pass along the internal carotid to the cavernous plexus, and thence via the ophthalmic division of the 5th to the eyeball. They convey the impulses which cause dilatation of the pupil, and supply also the unstriped muscle in the insertion of the levator palpebrae into the upper lid. Paralysis of the cervical sympathetic is recognized by the following signs: apparent enophthalmos; slight drooping of the upper lid, due to paralysis of the unstriped muscle-fibres contained in it; contraction of the pupil with absence of dilatation on shading the eye or on instillation of cocaine; abolition of the cilio-spinal reflex; less commonly, absence of sweating, even after the use of pilocarpine, on the corresponding half of the head and neck, both in front and behind, extending as low as the 3rd rib and 3rd thoracic spine, and over the whole of the upper limb on the same side.

The changes associated with paralysis of the cervical sympathetic fibres are referred to as *Horner's Syndrome*.

## V MOTOR FUNCTIONS

To examine the motor functions of a patient, investigate five separate points:

1. The size of the muscles.
2. The state of muscular tone.
3. The muscular power.
4. The co-ordination of movement.
5. The presence or absence of involuntary movements.

### The Size of the Muscles

The size of muscles is most easily ascertained when they are firmly contracted, but may also be gauged by palpation. Wasted or atrophic muscles are not only smaller but also softer and more flabby than normal. When muscular wasting is accompanied by fibrosis the muscles feel hard and inelastic; they become shortened and it is not possible passively to stretch them to a normal degree. *Contracture* is then said to be present. Contractures are also caused by prolonged hypertonia in a group of muscles. Muscular atrophy is not only caused by neurological disorders. Generalized muscular wasting is seen in patients with carcinoma and other chronic diseases. Localized muscle atrophy may be due to injury or disease of a joint; this is well seen in the thenar muscles in association with arthritis of the 1st metacarpophalangeal joint or in the quadriceps in patients with affections of the knee-joint. In such instances muscular power is well preserved in relation to the degree of muscular wasting. Some patients with muscular dystrophy develop large muscles (pseudo-hypertrophy) due to pathological changes in the muscles themselves. The calves, buttocks and infraspinati are particularly affected. The enlarged muscles are weak in spite of their size.

### Muscular Tone

Muscular tone is the state of tension or contraction that is always found in healthy muscles. An increase in tone is spoken of as hypertonia and a diminution as hypotonia. The degree of tone is estimated by handling the limbs and moving them passively at their various joints. The maintenance of tone is dependent on a spinal arc, afferent fibres entering the spinal cord and connecting with the anterior horn cells, whence efferent fibres arise and pass to the muscles. Tone is diminished or lost if this reflex arc is affected by disease. Hypotonia therefore occurs in affections of the lower motor neurone or of the afferent sensory pathways (as in tabes).

Muscle tone is modified and regulated by impulses passing along the pyramidal and extra-pyramidal pathways. The cerebellum is also closely concerned with the maintenance of tone.

Tone may be increased in states of anxiety. It is reduced or lost in sleep and states of unconsciousness.

Hypertonia following lesions of the pyramidal system is termed spasticity and it has a characteristic distribution; the upper limbs being fixed in flexion and the lower in extension. It must be

remembered that plantar-flexion of the foot is physiological extension. The resistance of the hypertonic muscles sometimes disappears suddenly on passive movement, the so-called 'clasp-knife' spasticity. When spasticity results from a partial lesion of the higher motor pathways in the spinal cord the lower limbs are paralysed in extension (*paraplegia in extension*). If the disease progresses and the lesion becomes complete the lower limbs adopt an attitude of flexion (*paraplegia in flexion*); this is the result of the uninhibited action of the spinal flexor reflex.

Hypertonia resulting from disease of the basal ganglia is termed rigidity. The hypertonia is more uniform, but often it is so distributed as to produce a general attitude of flexion of the limbs and trunk, e.g. paralysis agitans. The resistance to passive movement is fluctuant, hence the use of the descriptive term 'cog-wheel' rigidity. Occasionally a plastic type of rigidity is found with lesions of the mid-brain or in catatonic states. The resistance to passive movement is steady and continuous. In hysterical rigidity the muscular resistance increases in proportion to the effort made by the observer to move the limb. The muscles usually feel firmer than normal in hypertonia.

When the muscles are hypotonic, passive-movement encounters little or no resistance, and when the limb is handled or shaken the unsupported part flops about inertly. Hypotonic muscles are abnormally soft to palpation. The outstretched, hypotonic upper limb usually shows an abnormal posture as in the cerebellar disorder of chorea. It is hyperextended at the elbow, the forearm is overpronated, the wrist being unusually flexed, and the fingers overextended at the metacarpo-phalangeal joints.

### The Muscular Power

Determine whether the patient is capable of performing gross muscular movements. Can he walk? Can he sit up in bed? Can he move each of his limbs as a whole?

Then investigate the range of the movements that the patient can make, and the strength of the principal muscles and groups of muscles separately.

The general rule in this investigation is to ask the patient to throw into action the particular muscle or group of muscles which one wishes to test, while the observer offers to that action a greater or less degree of passive resistance. The following is the method of procedure:

**Upper Limb**

*Abductor pollicis brevis.* This is an important muscle clinically as it is the only intrinsic muscle of the hand invariably supplied by the median nerve which is easily tested. The patient is asked to abduct his thumb in a plane at right angles to the palmar aspect of the index finger. The muscle can be seen and felt to contract.

*Opponens pollicis.* Ask the patient to touch the tip of his little finger with the point of his thumb.

*First dorsal interosseous.* Ask the patient to abduct his index finger against resistance.

*Interossei* and *lumbricales*. Test the patient's ability to flex his metacarpo-phalangeal joints and to extend the distal phalanges. The interossei also adduct and abduct the fingers. When these muscles are paralysed and power is retained in the long flexors and extensors of the fingers the claw-hand or *main-en-griffe* deformity is produced. The first phalanges are overextended and the distal two are flexed. The fingers are slightly separated.

*Flexors of the fingers.* Ask the patient to squeeze your fingers. It is advisable to present him with two fingers only, the middle finger being abducted to lie across the index, or trauma to the examiner may result.

*Flexors of wrist.* The hand being held with the palm upwards, ask him to bring the points of his fingers towards the front of the forearm.

*Extensors of wrists.* The hand being held with palm downwards, the observer grasps the patient's wrist and asks him to bend the hand up backwards as far as possible. The fingers should be at the same time held flexed, as the wrist can be extended by contraction of the long extensors of the fingers. If he is unable to produce dorsiflexion of the wrist, some weakness or paralysis of the extensors is present.

Slight weakness of the extensors of the wrist may be elicited by asking the patient to grasp something firmly in his hand. If the extensors are weak the wrist becomes flexed as he does so, owing to the flexor muscles getting the better of the extensors.

Weakness or paralysis of the extensors of the wrist leads to the condition known as *wrist-drop*.

*Brachio-radialis.* Place the arm midway between the prone and supine positions; then ask the patient to bend up the forearm whilst the observer offers opposition to the act by grasping the hand.

If the muscle is healthy, it will be seen and felt to stand out prominently at its upper part.

*Biceps.* The patient's elbow being held against his side, ask him to bend up the forearm while opposition is offered by grasping the hand or wrist. If the biceps is healthy, it will be observed to stand out prominently as it contracts.

The *triceps* is tested by asking the patient to straighten out his forearm whilst the observer endeavours to keep it flexed by means of passive resistance.

*Supraspinatus.* Ask the patient to lift his arm straight out at right angles to his side. The first 30° of this movement are carried out by supraspinatus. The remaining 60° is produced by the —

*Deltoid*, whose anterior and posterior fibres help to draw the abducted arm forwards and backwards respectively.

*Infraspinatus.* The patient is asked to tuck his elbow into his side with the forearm flexed to a right angle. He is then instructed to rotate the limb outwards, the elbow being held against the side throughout. The muscle can be seen and felt to contract.

*Pectorals.* Ask the patient to stretch his arms out in front of him, and then to clap his hands while the observer endeavours to hold them apart. Note whether both heads of the muscle are thrown into contraction or not.

*Serratus anterior.* When this muscle is paralysed the scapula is 'winged', the vertebral border projecting. The patient is unable to elevate his arm above a right angle, the deformity becoming more apparent as he tries to do so. Pushing forwards with the hands against resistance also brings out the deformity.

*Latissimus dorsi.* Ask the patient to clasp his hands behind his back while the observer, standing behind the patient, offers passive resistance to the downward and backward movement; or grasp the two posterior axillary folds and ask the patient to cough. In health the latissimus can be felt to contract.

**Trunk muscles.** Weakness of the muscles of the abdomen is shown by the patient's inability to raise himself in bed without the aid of his arms. Babinski's 'rising-up sign' consists in making the patient lie on his back with the legs extended and rise up without using his hands. In organic spastic paralysis of a leg the affected limb will rise first, owing to the rigidity; but in functional paralysis this does not occur. Paralysis of a portion of the anterior abdominal wall can be detected by the displacement of the umbilicus that occurs when the patient

attempts to lift up his head from the pillow against resistance. With paralysis of the lower segment the umbilicus moves upwards, but when the upper segment is affected the umbilicus is pulled downwards. So also with unilateral paralysis the umbilicus is displaced by contraction of the unaffected muscle. To test the *erector spinae* and muscles of the back, make the patient lie on his face and try to raise his head from the bed by extending the neck and back. If the back muscles are healthy, they will be seen to stand out prominently during this effort.

The method of detecting paralysis of the *diaphragm* has already been described (p. 37).

The *trapezius* is tested in its upper part by asking the patient to shrug his shoulders while the observer tries to press them down from behind. In its lower part it can be tested by asking him to approximate the shoulder-blades.

**The head muscles.** The methods of detecting weakness or paralysis in the muscles of the head have been referred to in the section dealing with the investigation of the cranial nerves (p. 224).

**The lower limb.** *The intrinsic muscles of the foot* are not usually examined in any detail. When the interossei are weakened or paralysed a 'claw-foot', analogous to the 'claw-hand', may develop. Rarely this deformity occurs in patients with spastic paraplegia of very long duration.

*Dorsi-flexion* and *plantar-flexion* of the feet and toes are tested by asking the patient to elevate or depress the part against resistance.

*Eversion* and *inversion* of the foot should also be investigated by instructing the patient to turn the foot outwards or inwards against resistance.

*Extensors of knee.* Bend up the patient's knee, and then, pressing with your hand on the sole of his foot, ask him to try to straighten it out again.

*Flexors of knee.* Raise his limb from the bed, supporting his thigh with your left hand and his ankle with your right. Then ask him to try to bend his knee.

*Extensors of thigh.* The knee being extended, lift the patient's foot off the bed, and ask him to depress it against resistance. If the extensors of the hip are paralysed he will be unable to do so.

*Flexors of thigh.* The knee being extended, ask the patient to raise his leg off the bed.

The *adductors of the thigh* are tested by abducting the limb and then asking the patient to bring it back to the middle line while passive opposition is offered to the act. In a similar way the *adductors* are tested by bringing the limb across the middle line and then asking the patient to move it outwards again.

*Rotators of thigh.* With this lower limb extended on the bed, ask the patient to roll it outwards or inwards, whilst passive resistance is offered by grasping his foot.

If, on carrying out any of these tests, a muscle or group of muscles is found to have only a feeble power of contraction, *paresis* of it is said to be present. If no contraction is elicited at all, the condition is one of *paralysis*. Apparent weakness may be due to simultaneous contraction of opposing muscle groups. This can be detected by palpating the muscles at the same time as their power is tested. This type of weakness is found in hysteria.

The term *hemiplegia* is applied to a condition in which there is paralysis of one side of the face, and of the arm and leg on the same side. If the paralysis of the arm and leg is on one side, and that of some of the muscles supplied by the motor cranial nerves on the other, the condition is one of *crossed paralysis*. The term *paraplegia* is applied to a paralysis of the lower part of the body; the term *monoplegia* to a paralysis of one arm (which is therefore characterized as a *brachial monoplegia*), one leg (*crural monoplegia*), or one side of the face (*facial monoplegia*).

The detection of hemiplegia in a patient who is comatose is often a very difficult matter. However, if the paralysis is of recent onset, one can usually detect in such a patient a greater degree of *limpness* in the paralysed limbs. If his arm, for example, is raised from his side and allowed to drop, it falls, if it is paralysed, as if it did not belong to him; the sound arm also falls, but not in such an utterly limp fashion. The face is asymmetrical, the angle of the mouth more open on the paralysed side, and the affected cheek moves loosely outwards and inwards on respiration. The abdominal and tendon reflexes may be abolished on both sides, but an extensor plantar response is often obtained on the hemiplegic side.

### Myasthenic Weakness

This is weakness which becomes more marked after muscles have been exercised, and it occurs in myasthenia gravis. It is most commonly seen in the extra-ocular muscles and those concerned with speech and swallowing.

### The Coordination of Muscular Movement

By muscular coordination is meant the cooperation of separate muscles, or groups of muscles, in order to accomplish a definite act. If such cooperation is absent or imperfect, the performance of certain acts becomes difficult or impossible, and the condition is then said to be one of *incoordination* or *ataxia*.

The coordination of groups of muscles is the product of various factors, among the chief of which are the afferent impulses coming from the muscles that never reach consciousness and those on which the sense of position of the limbs depends; the state of tone of the muscles, and in some acts, perhaps, cutaneous sensibility. When incoordination is present it is not always easy to say which of these factors is at fault. The movements that constitute an act can be controlled and directed by vision, but sight itself is not concerned in the coordination of movements. When, however, there is loss of the sense of position, the sensory defect may be compensated by vision, and the disturbance of movement may become apparent only when the eyes are closed or bandaged, or in the dark. Such ataxia occurs typically in *tabes dorsalis*, when sense of position is diminished or lost in the lower limbs.

**How to test coordination.** (i) *In the upper limbs.* Ask the patient to touch the point of his nose first with one forefinger and then with the other; or ask him to bring the points of the two forefingers together. If he is able to succeed in these tests naturally and without making random shots, no incoordination is present. He may then be asked to perform the same actions with his eyes closed; any additional irregularity of the movements can be due only to disturbance of the sense of position.

Another good test in the upper limb is to ask the patient to thread a needle. In this case the eyes must, of course, be left uncovered.

(ii) *In the lower limbs.* If the patient is able to walk, a good test in the lower limbs consists in asking him to walk along a straight line—e.g. the edge of a carpet. If incoordination is present he will soon deviate to one side or the other.

If he cannot walk, ask him, as he lies in bed, to place one heel on the opposite knee, first with his eyes open and then when they are closed.

Another method is to leave the eyes open, and then ask him to

follow with his toe one's forefinger as it describes circles in the air. If he is able to describe the circles accurately his power of coordination is good.

*Romberg's sign* is often regarded as a special test for the coordination of the lower limbs, but though its presence is often evidence that the sense of position in these limbs is defective, it may also be positive when the patient's instability is due to some other cause, e.g. aural vertigo or a lesion of the cerebellum. The patient is made to stand with his feet close together, and if he can do so he then closes his eyes. If the sign is present, he begins at once to sway about or may even fall. To elicit slight degrees of the phenomenon, it may be necessary to make the patient stand on tiptoe with his knees bent. The essential feature of the sign is that the patient is more unsteady standing with his eyes closed than when they are open. In sensory ataxia, caused for example by tabes dorsalis, the patient is unable to maintain his attitude without the aid of vision on account of defective sense of position in the lower extremities.

A special sign for cerebellar ataxia is dysdiadokokinesia; it consists of inability to execute rapidly-repeated movements. In order to test for it the patient is asked to flex his elbows to a right angle and then supinate and pronate his forearms as rapidly as possible. All normal persons can do this at approximately the same rate, but, as a rule, slightly less rapidly with the left than with the right arm. When, however, dysdiadokokinesia is present the movements are slow, awkward and incomplete, and often become impossible after a few attempts.

### Abnormal Muscular Movements

These consist of involuntary muscular contractions of various sorts. The first thing to note is whether the movements are widespread or localized.

If they are confined to one part of the body, note the joints at which the movements occur, and the muscles or groups of muscles involved. The term *spasm* is often applied to any exaggerated and involuntary muscular contraction. The contraction may either be continuous, in which case it is said to be *tonic*; or there may be a series of short contractions with complete or partial relaxation of the muscle in the intervals, and in that case they are spoken of as *clonic*.

Tetanic spasm is observed in its completest form in tetanus, strychnine-poisoning, hydrophobia, and some kinds of hysterical

fits. It may lead to a bending of the whole body backwards (*opisthotonus*), or sideways (*pleurothotonos*), or forwards (*emprosthotonos*). The jaws may also be firmly clenched (*trismus*).

The term *tetany* is applied to a symptom-complex occurring under widely varying circumstances. The underlying disturbance is a diminution in the ionized calcium in the serum. There is a resultant hyper-excitability of the neuro-muscular apparatus which manifests itself by intermitting spasms of the muscles.

These spasms may affect any muscles of the body, but they most commonly occur in the periphery of the limbs. They are usually bilateral, in severe cases painful, and they may last for ten to twenty minutes and recur over many weeks.

An attack begins with a sensation of tingling and stiffness in the fingers. The thumb is forcibly adducted, the fingers pressed closely together, being flexed at the metacarpo-phalangeal joints and extended at the interphalangeal joints, sometimes the index finger is more powerfully flexed than the other fingers, the palm of the hand is made hollow by the approximation of its outer and inner margins, the whole hand assuming a conical shape—‘accoucheur hand’ (Plate V). In severe cases the wrist and elbows may be flexed and the shoulders adducted. When the lower limbs are affected the toes and ankles are plantar-flexed, the soles of the feet hollowed out, and the knees and hips extended.

Laryngospasm may occur in tetany. In rickets it is especially common and often appears without the occurrence of tetany. It is commonly termed ‘*laryngismus stridulus*’. At any time in the night or day the child affected will hold its breath until the face is cyanosed. Then the momentary spasm of the glottis relaxes, and as it does so air is drawn past the still closely approximated vocal cords with a high-pitched crowing sound.

In tetany the muscles of the trunk are involved only rarely, those of the face are sometimes affected, the lips being pursed up—‘carp mouth’—and spasm of the oculo-motor muscles sufficient to cause diplopia is occasionally seen.

The following physical signs peculiar to tetany are important, the more so since they often persist between attacks:

*Trousseau's sign.* Pressure upon the vessels or nerves of the limb, for example by a tourniquet or sphygmomanometer bag, will produce the typical spasm (‘accoucheur hand’) or will augment it if it is already present.

*Chvostek's sign.* Tapping over muscles and over nerves super-

ficially situated induces spasm. For example the light tap of a patella hammer over the facial nerve in front of the lobe of the ear causes muscular twitchings over the whole of that side of the face.

*Erb's sign.* Owing to increased electrical excitability of the motor nerves, fibrillation and spasm may be induced in the muscles by cathodal stimulation with currents subminimal to the normal subject.

*Occupational cramps* may occur in persons whose occupations involve complicated movements of the fingers for long periods of time, for example, telegraphists, clerks and violinists. They are psychogenic.

*Conjugate ocular spasms* or 'oculo-gyric crises' sometimes occur as a sequel of encephalitis lethargica. They consist of attacks of spasmodic conjugate ocular deviation usually upwards, which last about half an hour. During the attack, the patient is unable to deviate the eyes downwards below the horizontal plane, and any attempt at downward displacement is associated with intense tremor of the lids.

Muscle spasms with *epileptic attacks* may be tonic or clonic, and require careful description. If the muscular contractions are clonic and widespread, they are termed *convulsions*. The important features of the history of a patient with fits have been mentioned on p. 9. Should the observer be fortunate enough to witness an attack, he should note:

i. *The nature and distribution of the movements.* Are they general, or confined to one limb or part of a limb? What part is first and what last affected? Are the convulsions tonic or clonic? Is there any struggling, arching of the back, or attitudinizing? Are the abdominal muscles involved or not?

ii. Is there any *involuntary evacuation* of the bladder or rectum? Is there any blood or froth about the mouth? Does the patient change in colour?

iii. *The state of the eyes.* Are the eyes open? Is the corneal reflex present or abolished? Do the pupils react to light? Is there any incoordinate movement of the eyeballs?

iv. How does the patient behave *after the fit*?

If one group of muscles is first affected, the spasm spreading to others by degrees, it indicates a spread of the discharge along the cortex cerebri. This occurs typically in Jacksonian epilepsy.

*Myoclonus* is the term given to shock-like contractions occurring in individual muscles. They have been observed in certain forms of

encephalitis and are also encountered as manifestations of minor epilepsy. Their frequency varies from 10 to 80 contractions a minute, but they usually have no definite rhythm.

*Tremor* consists of more or less rhythmical oscillations of a part or parts of a limb, and is due to the alternate contractions of a group of muscles and its antagonists. Tremor may be either *fine* or *coarse*. Fine tremor is usually more easily felt than seen. It occurs in exophthalmic goitre, alcoholism, and in some forms of metallic poisoning. All forms of tremor are most easily seen by increasing the leverage at which the affected muscles act. Thus, tremor of the upper limbs is often brought out by getting the patient to extend his arms in front of him. In describing tremor, always note whether it is constantly present, or if it is affected in any way by voluntary muscular action. Also observe its rate, the amplitude of the movements, and whether they are regular or irregular. Ask the patient to lift a glass of water to his lips, and note whether the tremor is increased thereby (as it is in cases of cerebellar ataxia), or whether it is diminished or altogether abolished.

Tremor which only comes on when the patient attempts to use the affected muscles is described as *intention tremor*.

Clonic contraction of bundles of fibres in a muscle is termed *fasciculation*. It is seen in many cases of progressive muscular atrophy, and indicates an abnormal state of nutrition in the anterior horn cells connected with the affected fibres. The term *fibrillation* is reserved for contractions of individual muscle fibres. Such movements cannot be detected clinically, but may be recorded by the electromyograph.

To the transient flickering of a few muscle-fibres (commonly known as 'live-flesh' or 'live-blood') the term *myokymia* is applied. It is most often seen in the orbicularis palpebrarum, and is usually an indication of fatigue or debility. It also occurs as an independent condition, and is then more or less general.

The term *choreiform* is applied to involuntary movements of a non-purposive character occurring in individual muscles or groups of muscles. Such movements are seen most typically in chorea or St Vitus's dance. They consist of abrupt involuntary twitchings or contractions which cause the patient (usually a child) to seem fidgety and unsettled. They are increased by mental agitation, but are often diminished by voluntary muscular effort.

If the movements are limited to one side of the body the term *hemichorea* is applied.

Choreiform movements, if slight, can be elicited in two ways. First, one may ask the patient to hold both hands straight up above the head; or, second, one may ask him to spread out his hands, palms downwards, on the extended hands of the observer. In the former case it may be observed that the patient is unable to hold up his hands steadily for any length of time; in the latter, one may notice that little twitchy movements soon become evident in the patient's fingers.

If the patient is unable to write, one may get him to scrawl his name with the affected hand, and keep the result for purposes of comparison later. In this way one is able to estimate any increase or diminution in the choreic movements.

Choreiform movements also occur as a result of cerebral disease, they are then usually limited to one side of the body, and as they appear after local lesions they are sometimes described as *post-hemiplegic chorea*. Hemichorea without hemiplegia is seen in lesions of the corpus luisii.

*Tics* are coordinated, repetitive, purposive acts which are started in the first place by some external cause, or by an idea. By repetition they become habitual and finally involuntary, without any relation to the cause that first excited them. They may assume various forms; perhaps the most common are blinking of the eyes, smacking the lips, or rotation or nodding of the head. They can be distinguished from other involuntary movements by their complexity, and by their always retaining their purposive character.

The term *athetosis* is used to describe slow muscular contractions which lead to continuous and deliberate twisting movements specifically affecting the hands and feet but sometimes extending to involve the whole limbs and even the trunk.

The last point to be noted regarding any abnormal muscular movement is whether or not it persists during sleep.

## VI SENSORY FUNCTIONS

In investigating the sensory functions of a patient, we have to test the acuteness of the following forms of sensibility:

1. Tactile sensibility. This includes the powers of appreciating light touch and pressure, and of tactile localization and discrimination.
2. Sensibility to pain.

- 3. Thermal sensibility.
- 4. The sense of position and the appreciation of passive movement.
- 5. The recognition of the size, shape, weight and form of objects.
- 6. The appreciation of vibration.

In addition, the presence or absence of any abnormal sensations is noted.

At the outset explain to the patient the nature of the tests to be performed and so secure as far as possible his intelligent cooperation. The eyes should then be closed, or the part under examination screened from sight, and the different forms of sensibility tested as follows:

**1 Tactile sensibility.** Use a wisp of cotton-wool or a fine camel-hair brush. If it is desired to test the sensibility of the skin to light touch over a hairy part, it is essential to shave it, as the sensibility of the hairs themselves is so acute.

Tell the patient to say 'Now' every time he feels a touch. Compare corresponding points on opposite sides of the body, and employ every now and then a negative test, asking the patient if he feels you touch him, in order to prevent his making random replies. The appreciation of *pressure touch* should then be tested; this may be done by touching him with the point of a finger or any blunt object. It is important that its temperature should not differ much from that of the skin, and the pressure must not be so heavy as to give pain or discomfort. Ask him also to localize the stimulus by describing or in other way indicating the exact position of the spot touched. This is important, as a patient may be able to feel the stimulus and yet not be able to localize it.

Sensibility to touch may be altered in various ways. (1) It may be entirely abolished. This constitutes *anaesthesia*. If the abolition affects the whole of one side of the body, it is termed *hemianaesthesia*. If the existence of anaesthesia is discovered, one must at once proceed to mark out its exact extent and boundaries. Partial loss of tactile sensation is called *hypoesthesia*. (2) Sensibility may be so altered that what should in health be felt as a mere touch produces a painful impression resembling pricking or burning. This is generally called *hyperesthesia*. Hyperesthetic spots are sometimes met with, especially in hysterical patients. The commonest sites for these are over the brim of the pelvis, in the inframammary region, along the

vertebral column, and on the scalp. Pressure on such spots may sometimes induce hysterical fits. (3) Sensation may be appreciated well enough, but there may be great delay in its conduction, an appreciable interval occurring between the application of the stimulus and the response of the patient. This *delayed conduction* exists not infrequently in cases of tabes. (4) The stimulus may be badly localized, the patient believing, for example, that the outer side of a limb was touched when the stimulus was really applied to its inner aspect. Sometimes a touch on one side of the body is referred to a corresponding point on the opposite side.

*Two-point discrimination.* Ability to discriminate between two points is tested by the use of dividers, or two pins. The patient is asked whether he is being touched with one or both points. Normally 1 cm. of separation of the points is appreciated on the palmar surfaces of the thumb and fingers, a wider separation being required on the forearm and lower limb.

**2 Sensibility to pain.** Pain may be evoked either by a cutaneous stimulus, as the prick of a pin, or by pressure on such deeper structures, as the muscles or bones. Sensibility to superficial and to pressure pain should be tested separately.

(a) *Superficial pain.* The point of a steel pin or needle may be used as the stimulus. Care must be taken that the patient distinguishes between the *sharpness* of the point (that is, its relative size) and the *pain* which the prick evokes; it often happens that even when sensibility to pain is abolished he can recognize that the stimulus is pointed, and thus confuse the observer by calling it 'sharp'.

(b) *Pressure pain* is examined by squeezing the muscles or the tendo Achilles. Abolition of pressure pain is often the most prominent sensory disturbance in tabes dorsalis.

Absence of sensibility to pain is termed *analgesia*; partial loss of pain sensibility is called *hypoalgesia*; and an exaggerated sensibility, so that even a mild stimulus causes an unnatural degree of suffering, is known as *hyperalgesia*.

**3 Thermal sensibility** is conveniently examined by using test-tubes containing hot and cold water. The part to be tested is touched with each in turn, and the patient says whether each tube feels hot or cold. It is often important to determine the thresholds for heat and cold, i.e. the lowest temperature that feels warm and the highest that is cold. This can be done by noting the temperatures of the water in

the tubes on thermometers contained in them; to do this, it is better to use large copper or glass test-tubes. Note also the reactions evoked by high and low temperatures and the sensations they produce in the patient. It frequently happens that such temperatures evoke only pain, and may be called indiscriminately hot or cold.

The different forms of sensibility already mentioned may have to be tested on mucous membranes as well as on the skin surfaces. The sensibility of some viscera is also important. Thus the absence of pain on squeezing the testicle may be an early sign of tabes.

**4 Sense of position.** The patient's eyes being carefully shut, take hold of one of his limbs and move it about in various directions through the air, finally leaving it in some definite position, say semi-flexed and slightly elevated; then ask him to put the corresponding limb in a similar position. If there is no paralysis of the latter, and yet the patient is unable to imitate with it the position of the other, then there is reason to believe that the sense of position is impaired.

In the case of the hand the patient may be told that the fingers of one hand will be moved, and that he must imitate with the other the position in which they have been placed. In the case of the foot he may be told that the great toe will be placed pointing upwards or downwards, and that he must try to tell which it is.

In testing a patient's sense of position in this manner, be careful not to allow the part tested to touch any other skin surface; otherwise the patient will be able to appreciate its position by the information derived from his ordinary sense of touch.

A very delicate test for the sense of position in the upper limbs consists in shutting the patient's eyes and then making him hold his arms straight out in front of him with the fingers in a horizontal row. After a moment or two, if the muscular sense is defective, the fingers cease to remain in an even line. Some will rise a little, others fall, or even become twisted in below the rest.

The *appreciation of movement* is closely related to the sense of position, and should be tested at the same time. Grasp any segment of a limb firmly, and then move it gradually into another position; ask the patient to say 'Now' as soon as he recognizes the movement, and note the angle through which the limb was moved. If the appreciation of movement is diminished this angle is many times greater than that which is necessary in a normal limb, but if the defect is slight it may be necessary to measure the range of the movement accurately for comparison. Movements of  $10^{\circ}$  can be appreciated at

all normal joints. Finally, test if the patient can recognize the direction of the movement, that is, whether the joint is flexed or extended. It often happens that the patient can recognize the occurrence of a movement though he is ignorant of its direction.

**5 The recognition of size, shape and form.** These faculties can be tested most accurately in the hands with the eyes closed. To test size, place in the patient's palm objects of the same shape, but of different sizes, as small rods or matches of different length. Two objects should be applied consecutively, and he is asked to say which is the larger.

To test the power of recognizing form, familiar objects, as coins, a pencil, a penknife, scissors, etc., are placed in the hand, and the patient is asked to identify them or to describe their form. Loss of this power is generally known as *astereognosis*.

**6 Appreciation of vibration.** If the foot of a vibrating tuning-fork is placed on the surface of the body the vibrations can be felt, provided they are sufficiently strong. This is a valuable test, as the ability to appreciate vibration may be lost in various diseases, as in tabes dorsalis, peripheral neuritis, and conditions that involve the posterior columns of the cord. A heavy tuning-fork of 128 vibrations a second is generally employed. Make the fork vibrate by striking its prongs gently on a firm object, and place its foot immediately on the part to be tested; ascertain if the patient perceives the vibrations, and if so, ask him to say at once when he ceases to feel them. If the fork is then transferred to the observer, it can be seen if the sensibility to vibration is diminished or not, and the amount of diminution can be measured by the further time that the vibration can be perceived by the normal parts. It should be noted that there is some loss of vibration sense in the feet and legs in old age.

**Are there any abnormal sensations present?** These are termed *paraesthesiae*, and consist in various sensations experienced by the patient in the absence of any outward stimulus. The commonest of these are a feeling of 'pins and needles', of numbness, of heats or chills, of pressure or tightness, of itching—sometimes termed *pruritus*—or a feeling as if insects were crawling over the body (*formication*).

**Sensory inattention.** This phenomenon is sometimes found in patients with lesions of the parietal lobes; it is demonstrated as follows. The patient is asked to close his eyes. Mirror points on opposite sides of

the body are then tested simultaneously with identical stimuli, either painful, tactile or thermal. The patient is asked to say which side is being stimulated. If there is sensory inattention he will fail to appreciate the stimulus on the affected side of the body, that is the side opposite to his lesion. Sensory inattention may be found in the absence of any formal sensory loss and is thus a valuable sign. *Visual inattention* is tested along similar lines, the examiner using simultaneous movements of his index fingers in opposite fields as competing stimuli. The patient with visual inattention fails to notice the movement of the finger in the affected field. This again may occur in the absence of any formal field defect.

## VII REFLEXES

There are three types of reflex:

1. The superficial reflexes.
2. The deep or tendon reflexes.
3. The organic reflexes (including the action of the sphincters).

### 1 Superficial Reflexes

Here the simplest form of reflex action is concerned. On stimulation of a certain part of skin or mucous membrane, contraction of certain muscles results. The path of the impulse is by the sensory nerve-fibres to the grey matter of the cord or to a higher centre in the brain-stem or forebrain, thence by motor nerve-fibres to the muscle. A lesion in any part of this path causes the reflex to disappear. Thus, anaesthesia of the skin, disease of the sensory fibres or posterior nerve-roots, changes in the grey matter of the cord, lesions of the motor nerve-fibres or of the fibres of the muscles, may all cause abolition of the superficial reflexes. In addition, the reflex excitability of some individuals is normally much greater than that of others, which makes it difficult to estimate the value of slight alteration in the reflexes unless the lesion is unilateral, in which case the healthy side can be taken as a standard of comparison. The investigation of the superficial reflexes is of value as affording information regarding the health of the reflex arc concerned, and as a guide to the presence or absence of disease elsewhere. In hemiplegia the superficial reflexes are disturbed on the paralysed side.

The chief superficial reflexes of spinal origin, their nature, the mode of obtaining them, and the level of the cord concerned in their production, are given in the table on p. 271.

## CHIEF SUPERFICIAL REFLEXES OF SPINAL ORIGIN

<i>Reflex</i>	<i>How excited</i>	<i>Result</i>	<i>Level of cord concerned</i>
<b>ANAL</b>	Stroking or scratching the skin near the anus	Contraction of the anal sphincter	3rd and 4th sacral segments
<b>BULBO-CAVERNOUS</b>	Pinching dorsum of glans penis	Contraction of bulbocavernosus	3rd and 4th sacral segments
<b>PLANTAR</b>	Stroking sole of foot	Movements of toes, of toes and foot, or leg	Lower part of lumbar enlargement (5th lumbar and 1st sacral segments)
<b>CREMASTERIC</b>	Stroking skin at upper and inner part of thigh*	Drawing upwards of testicle	1st and 2nd lumbar segments
<b>ABDOMINAL</b>	Stroking abdominal wall below costal margin, at level of umbilicus and in iliac fossa	Contraction of abdominal muscles	7th to 12th thoracic segments
<b>SCAPULAR</b>	Stroking skin in interscapular region	Contraction of scapular muscles	5th cervical segment to 1st thoracic

\* The cremasteric reflex can often be most easily elicited by pressing over the sartorius in the lower third of Hunter's canal.

### The Plantar Reflex

This reflex demands special consideration. To elicit it the muscles of the lower limb should be relaxed and care should be taken that the sole of the foot is warm. The outer edge of the sole of the foot is stimulated by gentle scratching with a Yale key or a pin. In healthy adults a minimal stimulus produces a contraction of the tensor fascia lata, often accompanied by a slighter contraction of the adductors of the thigh and sartorius. With a slightly stronger stimulus, flexion of the four outer toes appears, which increases with the strength of the stimulus till all the toes are flexed on the metatarsus and drawn together, the ankle being dorsiflexed and inverted. With still stronger stimuli, violent regular movements of the limb occur, which spread to the lower part of the trunk and to the opposite side.

It is doubtful whether the plantar reflex is ever completely absent in healthy subjects.

In infants between six and twelve months old the reflex is very brisk, and differs markedly from that in adults. The earliest response is in the great toe, which is drawn back. This is followed by extension and spreading out of all the toes, with eversion of the foot or dorsiflexion of the ankle, and subsequently by flexion of the hip and knee ('infantile response').

**Babinski's sign.** During sleep the plantar reflexes are diminished and the infantile and adult forms preserved, save in some children up to the age of 12 years, to whom in deep sleep the infantile form of reflex returns. In pathological conditions the reflex varies, and may be of great diagnostic importance. In *lesions of the pyramidal systems* an alteration in the response was first described by Babinski. In this, which is spoken of as *Babinski's sign*, or the 'extensor response', the reflex closely resembles that obtained in infants, but differs in a few points. The whole response is more deliberate than that obtained normally either in adults or infants, and appears with much more certainty than does the flexor response to each stimulation. Extension of the great toe precedes all other movement. It is followed by spreading out and extension of the other toes, dorsiflexion of the ankle, and flexion of hip and knee. The small amount of movement at the ankle is less conspicuous than the brisk movement in the normal response. The extensor response is most easily elicited by stimulation of the outer part of the sole, and, with slight pyramidal lesions, may be evoked from this region alone when a normal flexor response is

obtained by stimulating the inner part. If the lesion is progressive, the area in which the extensor plantar reflex can be excited (receptive field), increases, and spreads first inwards over the sole of the foot, and then upwards along the leg to the knee or even the groin. For this reason extension of the great toe, generally associated with some dorsiflexion of the foot, can often be obtained by squeezing the calf or pressing heavily along the inner border of the tibia (*Oppenheim's sign*), or by pinching the tendo Achilles (*Gordon's reflex*), when the upper motor neurones are injured or diseased. The extensor response is met with in adults only in cases of organic disease involving the pyramidal tract.

### The Superficial Abdominal Reflexes

These reflexes too are valuable signs, as they disappear when the pyramidal tract of the same side is in any way affected. The lower abdominal reflex is then abolished earlier than the upper. It is often impossible to obtain them in old or obese people, or in women who have borne many children.

The following superficial reflexes are dependent on cranial nerves:

**Conjunctival.** Elicited by touching the conjunctiva, resulting in contraction of the orbicularis palpebrarum. The nerves concerned are the 5th (sensory) and the 7th (motor).

**Pupil reflexes.** (See p. 241.)

**Corneal reflex.** Consists in rapid closure of the eyelids on touching the cornea (e.g. with cotton-wool). It depends upon the integrity of the first division of the 5th cranial nerve on the afferent side and upon that of both 7th cranial nerves on the efferent.

**Palate reflex.** Elevation of the palate on touching the mucous membrane covering it. The nerves concerned are the trigeminal and glosso-pharyngeal on the afferent side and the vagus on the efferent.

### 2 Deep or Tendon Reflexes

If a muscle is put upon the stretch and its tendon is sharply struck, the muscle immediately contracts. This is spoken of as a deep or tendon reflex. The tendon reflexes are dependent upon the integrity of spinal reflex arcs consisting of afferent and efferent pathways. The

upper motor neurones exert an inhibitory effect on these reflex arcs and when this influence is cut off the tendon reflexes are increased.

Exaggeration of the tendon reflexes, unless some other disease coexists, is always associated with lesions of the upper motor neurones—i.e. with lesions affecting either the motor cortex or the fibres passing from it to the anterior horns of the cord. A similar exaggeration may be brought about by anything that stimulates the reflex arc, as strychnine, the toxin of tetanus, exposure to cold, and other factors. Attention or expectation can also induce a state of greater excitability, and the reflex response is greater if the muscles concerned are not fully relaxed; this is probably the explanation of the frequent exaggeration of these reflexes in hysteria and other functional conditions. Increase of the tendon reflexes is consequently not invariably a sign of organic disease.

On the other hand, anything that impairs the activity of the reflex arc makes it correspondingly difficult to elicit the tendon reflexes. Their diminution or abolition is therefore always associated with disease of the lower afferent and efferent neurones, or of the reflex centres in the grey matter of the spinal cord. Hence it is that in tabes dorsalis, in which the posterior roots are involved, and in peripheral neuritis, in which both motor and sensory fibres are generally affected, the deep reflexes are absent.

The tendon reflexes may be normal in cerebellar disease but sometimes they are altered, particularly the knee-jerk which is pendular in quality.

In a lesion—e.g. a fracture-dislocation—which produced complete transverse destruction of the cord at any level, one might expect that, owing to the cerebral influences being cut off, all the deep reflexes below that level would be exaggerated; but for the first few weeks, at least, all the reflexes are totally abolished. This seems to be due to a state of spinal shock in which the activities of the isolated portion of the cord are depressed as a result of its severance from the rest of the central nervous system. Later, however, if the general condition of the patient is satisfactory, the reflexes reappear, and a condition may develop in which a single stimulus is capable of producing a widespread effect. Thus, if the spinal cord is divided in the midthoracic region, stimulation of the abdomen or lower limbs may evoke a bilateral flexor spasm of these parts, along with reflex evacuation of urine and perhaps faeces, and an outburst of sweat from the lower limbs and trunk. This reaction has been called a 'mass-reflex'. But if bedsores, cystitis, or serious nutritional disturbances develop,

producing general debility, the reflexes may remain absent or disappear again.

### The Patellar Tendon Reflex

The knee-jerk or patellar tendon reflex is the best-known of the deep reflexes. It consists in a contraction of the quadriceps extensor when the patellar tendon is tapped. The spinal segments concerned are the 2nd, 3rd, and 4th lumbar. It is best tested with the patient supine. The examiner's hand is passed under the knee to be tested and brought to rest upon the opposite knee; the knee to be tested rests on the dorsum of the observer's wrist. Now distract the patient's attention in some way and strike the quadriceps tendon midway between its origin and insertion with a patellar hammer. Following the blow there will be extension of the knee from contraction of the quadriceps.

The briskness of the knee-jerk varies greatly in different individuals. In health it is hardly ever entirely absent. Sometimes one is unable to elicit it without applying *reinforcement*. This is done by asking the patient to make some strong voluntary muscular effort with the upper limbs; for example, to hook the fingers of the two hands together and then to pull them against one another as hard as possible. While he is doing this, a further attempt is made to elicit the knee-jerk which is often successful. Reinforcement acts by increasing the muscular tone throughout the body.

The following tendon reflexes are similar in nature to the knee-jerk, but they are connected with the spinal cord through anterior and posterior roots at different levels, and so are valuable in diagnosis.

### Ankle Jerk

Place the lower limb on the bed so that it lies everted and slightly flexed. Then with one hand slightly dorsiflex the foot so as to put the tendo Achilles on the stretch, and with the other hand strike the latter on its posterior surface. A sharp contraction of the calf muscles results. This reflex can also be conveniently elicited when the patient is kneeling on a chair. It depends upon the 1st and 2nd sacral segments.

### Triceps Jerk

Flex the elbow, then tap the triceps tendon just above the olecranon. The triceps contracts. The reflex depends upon the 6th and 7th cervical segments.

**Biceps Jerk**

The elbow is flexed to a right angle and the forearm placed in a semipronated position; the examiner than places his thumb on the biceps tendon and strikes it with the patellar hammer. The biceps contracts. The 5th and 6th cervical segments of the cord are concerned.

**Radial Supinator Jerk**

A blow upon the styloid process of the radius produces flexion of the elbow. This reflex also depends on the 5th and 6th cervical segments. With lesions at this level the reflex is abolished, but when it is tested flexion of the fingers may result from stretching of the flexor muscles in the forearm by the blow. This phenomenon is known as inversion of the radial supinator reflex.

**Jaw Jerk**

Ask the patient to open his mouth, but not too widely. Place one finger firmly on his chin and then tap it suddenly with the other hand as in percussion. A contraction of the muscles that close the jaw results. This jerk is sometimes absent in health, and is increased in upper motor neurone lesions above the 5th nerve nuclei.

**Clonus**

The phenomenon of clonus is often elicitable when the tendon reflexes are exaggerated as a result of a pyramidal lesion.

*Ankle-clonus.* Bend the patient's knee slightly and support it with one hand, grasp the fore part of the foot with the other hand and suddenly dorsiflex the foot. The sudden strain put upon the calf muscles causes them to contract. The pressure of the hand upon the sole of the foot is meanwhile continued, and when the contraction ceases causes the muscle again to become tense and so produces another contraction in the latter. In this way a whole series of contractions—i.e. a clonus—results.

The relative tendency to the development of ankle-clonus on the two sides is best estimated by slowly dorsiflexing the foot and observing the exact point at which the movements first begin. The less the degree of dorsiflexion required to produce the clonus the greater is the tendency to the development of the latter.

In cases of functional paralysis a spurious clonus may be elicited. It is usually ill-sustained and irregular in rhythm, and can be recognized by the feeling of voluntary contraction in the muscles, especially at the beginning of the clonus.

Sustained ankle-clonus is nearly always a sign of pyramidal disease. The spinal segments concerned in it are the 1st to the 3rd sacral.

*Patella-clonus.* In cases where the knee-jerk is exaggerated, one can sometimes elicit a patella-clonus by extending the patient's leg and then suddenly pushing down the patella towards the foot. If the pressure on the latter is continued, a series of clonic contractions of the quadriceps can in many cases be produced.

### 3 Organic Reflexes and Sphincters

This term includes the reflexes governing such processes as respiration, deglutition, micturition and defaecation. They depend upon complex muscular movements excited by increased tension in the wall of the viscus concerned, or, in the case of respiration, partly by stimulation of a centre in the medulla.

One should ascertain from the patient whether he has any difficulty in swallowing, noting especially whether there is any regurgitation of food through the nose. The function of *deglutition* does not usually require to be specially tested beyond the examination necessary to exclude the existence of an obstruction.

#### Defaecation

The patient should be questioned as to any difficulty in the act, and as to the presence of rectal sensation. Note also the occurrence or not of incontinence of faeces.

The reflex action of the *anal sphincter* may be tested by introducing the oiled, gloved finger into the anus, and noting whether contraction of the sphincter occurs with normal force, whether it is weak or altogether inactive, or whether any spasm is excited.

The activity of the anal-sphincter reflex may also be tested by pricking the skin in the neighbourhood of the anus. If the conditions are normal, a brisk contraction of the sphincter should immediately be visible. This depends upon the 4th and 5th sacral segments.

#### Micturition

The patient should be questioned as to difficulty or pain in the act (*see p. 8*). He should be asked whether bladder and urethral sensation are normal. Then note whether there is either *retention* or *incontinence* of urine. If there is incontinence, ascertain by the use of the catheter whether it is due to the *overflow* from a distended bladder, or whether it is a *reflex incontinence*—i.e. whether the bladder merely

fills up and then empties itself completely by reflex action. In another group of cases the patient feels the desire to micturate, and is unable to restrain the act, which takes place at once. This is spoken of as *precipitate micturition*.

The bladder receives a double innervation from the spinal cord: (1) sympathetic fibres from the 11th and 12th thoracic and 1st lumbar segments, by way of the hypogastric nerves; and (2) parasympathetic fibres from the 2nd, 3rd, and 4th sacral segments, by way of the pelvic nerves. The parasympathetic fibres are thought to be of major importance. Sensory impulses from the bladder and urethra are carried to the brain *via* the spino-thalamic tracts. The pathways concerned with the voluntary initiation of micturition probably run downwards from the motor cortex in the posterior marginal part of the lateral columns of the spinal cord. The rectum, along with the whole of the large intestine, is also innervated by the pelvic nerves from the 2nd, 3rd, and 4th sacral segments.

## VIII TROPHIC DISORDERS

In disease of the nervous system the nutrition of different tissues or organs may be impaired. The *bones* may become more brittle, or exhibit spontaneous fracture (osteopathies), or the *joints* may be the seat of painless effusion with or without atrophy or enlargement of the articular ends of the bones (arthropathies). In other cases the bones and joints are involved together (osteoarthropathies). More commonly the *skin* is the seat of change. It may exhibit an erythema, which may pass on to ulceration and the formation of bedsores at points of pressure; pigmentary changes may develop, or various eruptions—urticarial, vesicular, pemphigoid or herpetic—or the skin may be simply glossy. Perforating ulcers may appear, usually on the toes or soles of the feet, as in tabes, or there may be actual gangrene, or the development of painless whitlows. In other cases it is the epidermic appendages which especially suffer change, the hair falling out, or the nails becoming dry and brittle. Atrophy of the *muscles* is a common phenomenon.

## IX SIGNS OF MENINGEAL IRRITATION

### Neck Rigidity

To test for neck rigidity the examiner places his hand behind the patient's occiput and flexes the head so that the chin touches the

chest. Normally this movement can be carried out without pain. In meningeal irritation the test causes pain in the neck, sometimes radiating down the back, and the movement is resisted by spasm in the extensor muscles of the neck. Neck rigidity is also caused by diseases of the cervical spine. Head retraction is an extensive degree of neck rigidity.

### Kernig's Sign

Kernig's sign is tested by attempting to carry out passive extension of the patient's knee when his hip is fully flexed. The test causes pain and spasm of the hamstrings in meningeal irritation affecting the lower part of the spinal sub-arachnoid space. If Kernig's sign is positive in the absence of neck rigidity the inference may be drawn that the patient is suffering from spinal meningeal irritation.

These two tests depend upon the fact that stretching the spinal nerve roots in conditions of meningeal irritation causes a reflex muscular spasm. They are positive in meningitis, sub-arachnoid haemorrhage, but also in patients with 'meningism', a state of irritation of the meninges, seen most commonly in young children with acute specific fevers.

### Straight Leg Raising

This test is used in patients with sciatica. The sciatic nerve and its component roots are stretched by passively elevating the patient's extended leg with the examiner's hand, which is placed behind the heel. The movement is restricted by pain in conditions in which the nerve or, as more frequently happens, its spinal roots are involved.

## X ANCILLARY INVESTIGATIONS

The following special methods of investigation are in common use in neurological practice.

### Lumbar Puncture

This is a procedure requiring some experience and should be carried out under supervision in the first instance. It is performed as follows:

Draw a line with a swab dipped in alcoholic solution of iodine vertically down the vertebral spines and another horizontally at the level of the highest points of the iliac crests. The lines intersect at the space between the 3rd and 4th lumbar spines or sometimes at the tip

of the 4th lumbar. The puncture may be made through either the 3rd or 4th interspace. The patient should be lying on his side on a firm couch, with the knees and chin as nearly approximated as possible. His back should be right at the edge of the couch and it is important that its transverse axis, i.e. a line passing through the posterior superior iliac spines should be quite vertical. Local anaesthesia may be produced by injecting 2 per cent sterile procaine, first raising a bleb under the skin, and, when this is insensitive, thrusting the needle in towards the centre of the intervertebral space, injecting the solution as one does so. A special platinum-iridium or nickel needle about 8 cm. in length should be employed (steel is too brittle); it should be of fine calibre and provided with a bevelled end and stylet.

Push the needle firmly through the skin in the middle line or just to one side of it and press it forwards and slightly upwards, the bevel pointing towards the side on which the patient is lying. When the needle is felt to enter the spinal cavity the stylet is withdrawn and the fluid which escapes collected in sterilized test-tubes. The puncture is sealed with collodion, and the patient should lie flat for 24 hours afterwards. The fluid should be collected in two test-tubes. If any blood is present, a marked difference in the amount in the first and second tubes indicates that the blood is due to trauma from the puncture.

It is an advantage to have a manometer connected with the needle, so that the pressure of the fluid can be measured at the time of puncture. If this is done, the patient's head must be on the same level as the sacrum and he must be breathing quietly and with his muscles relaxed. The normal pressure is from 60 to 150 mm. of fluid.

*Queckenstedt's test* is used to detect a block to the circulation of fluid in the spinal cord. With the needle and manometer in position and the patient breathing quietly as described, an assistant compresses both jugular veins. This causes a sudden increase in intra-cranial pressure, which is immediately seen in the manometer as a sudden rise of cerebro-spinal fluid pressure, followed by an equally rapid fall when the pressure on the jugular is released. A similar sudden rise and fall is seen if the patient is asked to cough. With slight degrees of blocking there may be a rise of pressure in the manometer followed by a very slow fall when the pressure on the jugular is released; and with more severe blocking no rise of pressure will be seen when the jugulars are compressed.

Much the commonest cause of a 'dry-tap', the failure to obtain

fluid, is an incorrectly performed puncture, and this is usually due to the patient not being in the correct position, so that the needle is not introduced at right angles to the transverse axis of the back, and misses the spinal canal. Occasionally, however, a 'dry-tap' is due to a complete blockage of the spinal canal, and under these circumstances it may be necessary to resort to *cisternal puncture* to obtain a specimen of cerebro-spinal fluid. This operation presents no great technical difficulty, but should only be undertaken when lumbar puncture attempted by a competent operator has been unsuccessful, and when there is no reason to suspect a tumour or abscess of the posterior fossa or a great increase of intracranial pressure. The patient's head is shaved for a few inches at the back. He is seated and his head is held well flexed between the hands of an assistant. The operator then finds the highest palpable spinous process, which is that of the second cervical and injects some 2 per cent procaine into the skin half an inch above this process. A lumbar puncture needle is introduced through this point, and passed forwards in a plane which passes through the point of introduction, the external auditory meatus and the nasion. At a point about 3 cm. from the surface, the resistance of the posterior occipito-atlantal ligament is felt. The needle is then introduced a little farther and should enter the cisterna magna. The medulla lies at a depth of about 3 cm. in front of the posterior occipito-atlantal ligament. A piece of adhesive tape wound around the needle 4·5 or 5 cm. from its point will prevent any danger of the medulla being damaged.

Neither lumbar nor cisternal puncture should be performed in patients with papilloedema, nor in patients otherwise under suspicion of having a cerebral tumour, until a neuro-surgeon has been consulted. Lumbar puncture should also be avoided in patients with disseminated sclerosis, if the diagnosis can be made without it.

**Examination of the fluid.** Normal cerebro-spinal fluid is clear and colourless like distilled water, with a specific gravity of 1006. Any yellowness of tint is pathological and is due either to old haemorrhage, obstructive jaundice or excess of protein. In *Froin's syndrome* a pronounced yellow colour (xanthochromia) is associated with great excess of protein and massive coagulation of the fluid. Formation of a clot in a colourless fluid indicates meningitis, usually tuberculous, or high protein from other cause, e.g. tumour or polyneuritis.

**Turbidity** of the fluid may be due to pus or to red blood-corpuscles. If it does not clear on standing it is due to micro-organisms.

The presence of *blood* may be due to injury to a vessel by the needle or to subarachnoid haemorrhage. In the latter case the blood is more uniformly mixed with the fluid, and the supernatant fluid, after the corpuscles have settled, is yellow.

**Cytology** of a turbid fluid is of great importance. A centrifugal deposit should be examined with Leishman's stain in order to obtain an idea of the character of the cells present.

To carry out a *cell-count*, take a capillary pipette and make a mark on it with a grease pencil about 3 cm. from its distal end. Using this marked-off portion as a unit of volume, take 4 volumes of cerebro-spinal fluid and one volume of any simple stain (Loeffler's methylene blue or carbol thionin). Mix well in a watch-glass or clean test-tube and place a suitably sized drop in a Thoma-Zeiss counting-chamber.

Adjust the draw-tube of the microscope so that the diameter of the field with the  $\frac{1}{2}$  in. objective is 8 small squares. Count the cells in 100 fields, refilling the chamber when necessary. Then the number of cells in 100 fields = the number in 1 c. mm.

For the area of one field = 50 small squares (approximately)

$$50 \text{ small squares} = \frac{50}{4000} \text{ c. mm.} = \frac{1}{80} \text{ c. mm. but the fluid is diluted to four-fifths strength, therefore}$$

$$100 \text{ fields} = \frac{100}{80} \times \frac{4}{5} \text{ c. mm.} = 1 \text{ c. mm.}$$

A special counting-chamber (e.g. the Neubauer) may also be used. It should be noted that a cell-count must be done immediately the fluid has been collected. Counts done some hours later give inaccurate results owing to the fact that pus cells stick together and to the sides of the tube, while endothelial cells break up in a short time. If any clot has formed an accurate cell-count cannot be obtained. Normal fluid contains up to 5 lymphocytes per c. mm.

An excess of cells ('pleocytosis') is described as being of the polymorphonuclear type if these cells are above 75 per cent of the total, and of the lymphocytic type if more than 90 per cent are lymphocytes. A mixed type also occurs in which the polymorphs amount to from 15 to 70 per cent of the total. The coccal forms of meningitis are associated with the polymorphonuclear type, virus meningitis and syphilis with the lymphocytic type, and tuberculous meningitis and poliomyelitis with either a lymphocytic or a mixed type.

### Chemical Examination

Chemical tests provide further useful information. Normal cerebro-spinal fluid contains only a trace of albumin and hardly any globulin, the *total protein* being not more than 40 mg. per 100 ml. The protein content can be roughly estimated by placing 2 ml. of fluid in a test-tube and carefully running in an equal quantity of absolute alcohol down the side. In normal fluid the line of junction is just visible; if protein is present in excess there is a turbid ring. The total protein is estimated more accurately by precipitation and comparison with standards.

In certain pathological conditions the globulin fractions in the cerebro-spinal fluid are altered. The *Lange* test takes advantage of this fact: varying dilutions of cerebro-spinal fluid are mixed in ten tubes with a colloidal gold suspension of constant strength. The degree of precipitation which results is expressed by arbitrary figures 0-5, 0 representing no change and 5 complete precipitation.

The *Wassermann* serological reaction is often performed on the cerebro-spinal fluid.

*Glucose* is present in normal cerebro-spinal fluid in a concentration of 50-75 mg. per 100 ml., which is less than the amount of sugar in the blood. If 1 ml. of fluid is boiled with 0.25 ml. of Fehling's solution the latter should be almost decolorized; if much blue is left it is an indication of reduced sugar content. Accurate estimation of the sugar is carried out in the same way as in blood. The amount of sugar is diminished in acute purulent meningitis.

The *chloride* content of the normal fluid is 720 to 750 mg. per cent as sodium chloride. Amounts below this are met with in cases of meningitis and above it in renal failure. The low chlorides in cases of meningitis (classically tuberculous) are more probably a measure of general chloride deficiency due to dehydration than any specific local effect of the organism. The chlorides are estimated by adding 2 ml. of fluid to 20 ml. of distilled water and titrating with standard silver solution, chromate of potash being used as indicator.

For a table showing the typical changes in the cerebro-spinal fluid in various diseases, see p. 284.

### Electro-encephalography

The electro-encephalogram (EEG) is a record of the electrical activity of the cerebral cortex. Electrodes applied to the patient's scalp pick up the small changes of electrical potential which are

## THE NERVOUS SYSTEM

TABLE SHOWING THE TYPICAL CHANGES IN THE CEREBRO-SPINAL FLUID IN VARIOUS DISEASES

	<i>Normal</i>	<i>Meningitis</i>	<i>Tuberculosis</i>	<i>Disseminated sclerosis</i>	<i>GPI</i>	<i>Tubes</i>	<i>Meningeal syphilis</i>	<i>Acute anterior poliomyelitis</i>
<i>Physical Characters</i>	Clear; colourless, no coagulum.	Meningo-coccal	Ginger-beer turbidity; coagulum	Colourless, with cobweb coagulum	Clear; colourless	Colourless; sometimes fine coagulum	Clear or turbid; sometimes fine coagulum	Usually clear; sometimes cobweb coagulum
<i>Cells—</i> Polymorphs Lymphocytes	0 to 5 } Cells per c.m.m.	200 to 2000 5 to 50	0 to 100 100 to 300	0 to 5 5 to 100	0 to 5 5 to 100	0 to 5 5 to 100	10 to 50 50 to 500	10 to 100 or higher; polymorphs at first. Lymphocytes later
<i>Total protein*</i>	20 to 40	50 to 200	50 to 200	30 to 60 rarely higher	40 to 100	30 to 60	50 to 200	30 to 60 in early days, rising to 100–200, and remaining high for 8 to 10 weeks
<i>Glucose*</i> Chondroes WR	50 to 75 720 to 750	0 to 15 600 to 680	15 to 50 480 to 550	Normal "	45 to 60 Normal +	45 to 70 Normal +	Normal +	100
<i>Culture</i>	Sterile	Meningo-cocci	TB in films in some cases, and may be cultured by special methods	Sterile	Sterile	Sterile	Sterile	—
<i>Lumbar curve</i>	0000000000 0000110000	0001244310	†	5555432100	0123210000	0123210000	0123210000	0123210000

• mg. per 100 ml.

† A paretic type of curve is found in 50 per cent of cases.

constantly taking place in the cortex. After amplification these changes are recorded on a strip of paper. Electro-encephalography is of particular value in the investigation of epileptic patients and in the localization of cerebral tumours and other expanding intracranial lesions.

### **Electro-myography**

This consists in recording the amplified electrical activity of muscle. The investigation is of assistance in the diagnosis of lesions of the lower motor neurone and of diseases of muscle.

### **Neuro-radiological Diagnostic Procedures**

Apart from the use of plain X-rays the following methods are often employed:

(a) **Air studies.** The ventricular system is outlined with air and radiograms of the skull are then taken. The size, shape, and position of the ventricles may be altered by expanding intracranial lesions. Atrophic changes in the brain may result in compensatory dilatation of a part of the whole of the lateral ventricles. The air may be introduced at lumbar or cisternal puncture (air encephalography) or by ventricular puncture (ventriculography); an equivalent volume of cerebro-spinal fluid must, of course, be withdrawn.

(b) **Cerebral angiography.** X-rays of the skull are taken immediately after the injection of a radio-opaque solution into the internal carotid or vertebral arteries. The injection is made percutaneously. This method is useful in demonstrating congenital vascular abnormalities, such as aneurysms and angiomas, and vascular tumours. Tumours may also be localized because they frequently cause displacement of the cerebral arteries.

(c) **Myelography.** This is used in the diagnosis of spinal compression. A radio-opaque substance is injected into the subarachnoid space, either by lumbar or cisternal puncture, after the removal of an equal volume of cerebro-spinal fluid. The substance, which is heavier than spinal fluid, is made to run up or down the spinal sub-arachnoid space by tilting the patient on a table under the X-ray screen. Partial or complete spinal obstructions can be demonstrated by this method.

## **10 EXAMINATION OF THE EYE, EAR, THROAT AND NOSE**

### **I THE EYE**

Before examining the various parts of the eye, one should test the visual acuity, and, when relevant, the colour sense and the extent of the visual fields.

#### **1 Visual acuity**

**(1) Test for distant vision.** Visual acuity is measured with Snellen's test-types, a series of letters of varying sizes so constructed that the top letter is visible to the normal eye at 60 metres, and the subsequent lines at 36, 24, 18, 12, 9, 6, and 5 metres respectively. Visual acuity ( $V$ ) is recorded according to the formula  $V=d/D$ , where  $d$  is the distance at which the letters are read, and  $D$  that at which they should be read. The patient is normally placed at a distance of 6 metres from the test types ( $d=6$ ) and each eye is tested separately. The patient reads down the chart as far as he can. If only the top letter of the chart is visible, the visual acuity is 6/60. A normal person should be able to read at least the seventh line, i.e. a visual acuity of 6/6. A person with an uncorrected refractive error may have a subnormal visual acuity, and a rough estimate of his corrected visual acuity may be obtained by asking him to view the chart through a pin-hole aperture. If the visual acuity is less than 6/60, the patient is moved towards the test-types until he can read the top letter. If the top letter is visible at 2 metres, the visual acuity is 2/60. Visual acuities of less than 1/60 are recorded as '*counting fingers*' (CF), '*hand movements*' (HM), '*perception of light*' (PL), or '*no perception of light*' (no PL).

If the patient wears glasses, the type of lens he is wearing may be determined as follows. Hold the lens in front of the eye and look at an object through it. Then move the lens from side to side and watch the object. If the latter seems to move in the opposite direction to the lens, the lens is convex; if in the same direction, it is

concave. Patients with myopia use concave (diverging) lenses and those with hypermetropia convex (converging) ones.

In order to tell whether a lens is spherical or cylindrical, look at a straight object through it and then slowly twist the lens round. If the lens is cylindrical, the object will appear to take up an oblique position. Patients who are astigmatic need cylindrical lenses.

(2) **Test for near vision.** Visual acuity at the ordinary reading distance is assessed with reading test types of varying sizes, the notation being based on the printers' 'point' system. The smallest print used is N5. The near vision is recorded as the smallest type which the patient can read comfortably.

### 2. Colour sense

This is most easily tested by the use of pseudo-isochromatic plates, the best-known being those of Ishihara. People with defective colour vision confuse certain colours. Pseudo-isochromatic plates are so constructed that a person with normal colour vision will read one number on a plate, while a person with defective colour vision will read a different number on the same plate.

The most common anomalies of colour vision are various types of red-green deficiency, inherited as sex-linked recessive conditions, which occur in about 8 per cent of males and 0·5 per cent of females in this country. People with blue-yellow deficiencies and with total colour-blindness are rare.

### 3. Visual fields (see p. 226)

#### Examination of the Eye

After the visual functions have been tested, the eyes should be examined systematically. The shape and position of the eyelids should be noted. Mongolian races have a long narrow palpebral aperture with an upward and outward obliquity and a characteristic fold of skin along the upper lid. The highest point of the aperture is at the junction of its middle and inner thirds, whereas in mongolism (Down's syndrome) the oblique palpebral aperture is short and wide with its highest point at the centre of the lid (Plate I).

*Ptosis*, or drooping of the upper lid, may be congenital or acquired. A congenital ptosis may be unilateral or bilateral, whereas an acquired ptosis is usually unilateral if due to paralysis of the third cranial nerve (p. 231) or of the cervical sympathetic (p. 253), and usually bilateral if due to a myopathy.

## 288 EXAMINATION OF EYE, EAR, THROAT, NOSE

*Lid retraction* is present if a band of white sclera is visible above the iris when the eyes are looking straight ahead. Lid retraction, which is usually due to thyrotoxicosis, is often associated with infrequent blinking and with lid-lag, i.e. the upper lid seems to lag behind the eyeball when the patient looks downwards. Patients with thyrotoxicosis frequently also have a slight degree of forward displacement of the eyeball (exophthalmos). Some patients with thyrotoxicosis, or some whose thyrotoxicosis has been successfully treated medically or surgically, develop a more severe and progressive form of exophthalmos, which may be associated with ocular palsies. Exophthalmos also results from space-occupying lesions in the orbit, and an apparent exophthalmos may be present when the eye is longer than normal, as in myopia.

The presence of an inflammation of the margins of the lids (marginal blepharitis) should be noted, together with any abnormality in the position of the lid margins, i.e. eversion (ectropion) or inversion (entropion) of the lashes.

*The lacrimal gland* is examined by pulling up the outer part of the upper lid while asking the patient to look downwards and inwards. Acute inflammations (dacryoadenitis) result in a tender swollen gland with oedema of the upper lid and localized conjunctival injection. Chronic dacryoadenitis, a painless enlargement of the lacrimal gland which is frequently bilateral, occurs in the reticuloses, sarcoidosis, and tuberculosis. Tumours of the lacrimal gland produce a hard swelling of the gland associated with proptosis.

*The conjunctiva* lining the eyeball (bulbar conjunctiva) and that lining the inner surface of the eyelids (palpebral conjunctiva) should be examined. In order to examine the palpebral conjunctiva of the lower lid, the lower lid should be pulled down while asking the patient to look upwards. To expose the palpebral conjunctiva of the upper lid, ask the patient to look downwards, then place the right thumb at the upper part of the upper lid and pull it upwards so as to evert the eyelashes. Grasp the lashes between the forefinger and thumb of the left hand and evert the lid by rotating it round the right thumb. The conjunctiva may be pale in anaemia, jaundiced, or injected in conjunctivitis. Marked injection of the bulbar conjunctiva with a mucopurulent discharge suggests a severe bacterial inflammation; marked injection with a little serous discharge is indicative of a viral infection; slight oedema of the conjunctiva with a milky hue suggests an allergic condition. Follicles on the upper palpebral conjunctiva occur in trachoma, whereas

their presence on the lower palpebral conjunctiva suggests an allergic condition or a conjunctivitis of viral origin.

In *conjunctivitis* the injection is maximal in the fornices (the junction of bulbar and palpebral conjunctiva), and this appearance must be distinguished from the circumcorneal injection that occurs in keratitis, anterior uveitis and acute glaucoma. In circumcorneal injection there is a narrow band of dilated blood vessel around the limbus, and the injection is minimal in the fornices.

*Inflammations of the cornea* (keratitis) may be superficial or deep, and are accompanied by circumcorneal injection. Superficial keratitis and corneal ulcers result in breaches in the corneal epithelium, and these breaches will stain with fluorescein. A drop of fluorescein is instilled into the conjunctival sac and the excess dye is then washed out with normal saline. Breaches in the corneal epithelium are stained green. Deep keratitis results in a hazy cornea, often with an intact epithelium; it is usually caused by a viral or syphilitic infection. Keratitis, or trauma to the cornea, may result in corneal opacities; small opacities are described as nebulae, larger ones as leucomata.

*Arcus senilis* is a crescentic opacity near the periphery of the cornea. It usually starts at the lower part of the cornea, extending to form a complete circle. It is common in old people, but may occur in the young (*arcus juvenilis*). It does not appear to have any significance.

In *anterior uveitis* (iritocyclitis) circumcorneal injection also occurs. In addition, white specks may be visible on the posterior surface of the cornea (keratic precipitates); there may be an exudate in the anterior chamber, and the pupil may be constricted and irregular due to the formation of adhesions between the edge of the pupil and the anterior surface of the lens (posterior synechiae). Other abnormalities of the pupils are described on p. 241.

The *ocular tension* may be roughly assessed by palpating the eyeball, although even with practice only gross variations from normal can be appreciated. The sclera is palpated with the two fore-fingers through the upper lid with the patient looking downwards, the other fingers resting on the patient's forehead. The degree of fluctuation gives an indication of the ocular tension. More accurate measurements of ocular tension can be made with Schiøtz or applanation tonometers. A diminished tension occurs in diabetic coma and in severe dehydration from any cause. A myopic eye frequently feels softer than a normal one. An increased ocular tension occurs in glaucoma.

### The Fundus

Examination of the fundus of the eye with an ophthalmoscope is an essential part of every complete medical examination. Valuable information may be obtained as to the state of the optic nerve head and of the arteries and veins of the retina, in addition to the detection of local ophthalmic conditions.

In routine medical examinations it is usually possible, with practice, to examine the optic disc and surrounding retina without dilating the pupil, but for a complete examination of the fundus the pupils should be dilated by instilling a few drops of 1 per cent cyclopentolate (Mydrilate) or 2 per cent homatropine into the conjunctival sacs. Patients with a predisposition to closed-angle glaucoma may have an attack of acute glaucoma precipitated when the pupils are dilated. Before a mydriatic is instilled, the patient should be asked whether he has ever seen haloes (coloured rings) around lights and, if he has, this, or the presence of a shallow anterior chamber, is a contra-indication to the use of a mydriatic. After the examination of the fundus has been completed, the effects of the mydriatic should be counteracted by the instillation of a few drops of 2 per cent pilocarpine.

The patient should be examined either sitting or lying down in a darkened room. He is asked to look straight ahead and to keep his eyes as still as possible. The ophthalmoscope is held a few inches from the patient's eye, and a suitable plus lens is used in the ophthalmoscope so that the iris is in focus. Opacities in the media of the eye (cornea, anterior chamber, lens, vitreous) will appear as black specks or lines against the red reflex of the fundus.

The ophthalmoscope should then be brought as close as possible to the patient's eye and the light directed slightly nasally. In this way the optic disc can be found, and in addition the light will not shine directly on the macula. If the patient's pupils are not dilated, shining a light on to the macula will make the pupils contract and may make the examination of the fundus difficult or impossible. If the optic disc is not in focus, the strength of the lenses of the ophthalmoscope should be gradually reduced until the disc becomes sharply focused. If the observer's eye is emmetropic and his accommodation is relaxed, the strength of the lens necessary to bring the fundus into focus gives an indication of the refractive error of the patient's eye. Plus lenses indicate hypermetropia, and minus ones myopia. The optic disc, the retinal blood vessels, the

macular region, and the periphery of the fundus should be examined in turn.

**1 The optic disc.** (1) *Shape.* The normal disc is round or slightly oval. If astigmatism is present, the disc may appear more oval than normal.

(2) *Colour.* The normal disc has a pale pink colour, distinctly paler than the surrounding fundus. The temporal side of the disc is usually paler than the nasal side.

In atrophy of the optic nerve, the disc becomes very pale and may even become white or greyish-white in colour. In oedema of the optic nerve-head, resulting from raised intracranial pressure (papilloedema) or from inflammation (papillitis), the disc is pinker than normal and may approach the colour of the surrounding retina. In pseudoneuritis, a congenital anomaly usually associated with hypermetropia, the disc may appear swollen and pinker than normal, but the retinal blood vessels are normal in appearance, corrected vision is normal and the condition is stationary.

(3) *Physiological cup.* In its central part there is usually a depression in the disc, the physiological cup. The cup is paler than the surrounding disc, and from it the retinal vessels enter and leave the eye. In glaucoma the cup may be greatly increased in size, and the retinal vessels will kink as they cross the edge of the disc. When the cup is deep (in advanced glaucoma), retinal vessels disappear as they climb from the floor to the rim, and reappear again as they bend sharply over the edge of the cup (Plate XXI).

(4) *Edge of the disc.* This is normally well-defined. In normal eyes there is sometimes a white scleral ring, a dark pigmented ring, or a stippled choroidal ring surrounding the optic disc.

**2. The retinal blood vessels.** These radiate from the disc, dividing dichotomously into many branches as they pass towards the periphery of the retina. The retinal arterics are narrower than the veins, are a brighter red in colour, and have a brighter longitudinal streak where light is reflected from their convex walls. Spontaneous retinal artery pulsation is an abnormal finding, and occurs in some cases of glaucoma and aortic regurgitation. Spontaneous venous pulsation is frequently seen in normal eyes; it never occurs in papilloedema. It is important to study the points where arteries and veins cross. Most frequently it is the artery that crosses the vein, and in normal eyes neither vessel shows any change in colour, diameter or direction.

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**3 The macular region.** This is situated about 1·5 disc diameters from the temporal border of the optic disc. It is recognized by being darker in colour than the surrounding fundus, and is frequently surrounded by a halo or annular light reflex. At the centre of the macular region is a small depression, the fovea, which is lighter in colour and often glistens. Pathological changes in the macular region are important, as they produce a greater reduction of vision than similar changes in any other part of the fundus.

**4 The periphery of the fundus.** This area can be examined only if the pupil is dilated with a mydriatic. Certain disease processes start in this region, for example retinal tears and retinitis pigmentosa.

The following is a brief description of the chief changes met with in the fundus which are important medically:

**Papilloedema** (Plate XXI) is a passive oedema of the optic nerve-head, most commonly due to raised intracranial pressure. There is an absence of inflammatory changes, and frequently there is little or no disturbance of visual function. In the initial stages of the condition there is an increased redness of the disc with blurring of its margins, the blurring appearing first at the upper and lower margins, particularly in the upper nasal quadrant. The physiological cup becomes filled in and disappears and the retinal veins are slightly distended. Spontaneous pulsation of the retinal veins is absent.

As the condition progresses the disc becomes definitely swollen. In order to measure the degree of swelling of the disc, it is necessary to start with a high plus lens in the ophthalmoscope and reduce the power of the lens until the centre of the disc is just in focus. The retina, a short distance from the disc, is then brought into focus by further reduction of the power of the lens. This further reduction indicates the degree of swelling of the disc (3 dioptres is equivalent to 1 mm. of swelling).

If papilloedema develops rapidly, there will be marked engorgement of the retinal veins with haemorrhages and exudates on and around the disc, but with papilloedema of slow onset there may be little or no vascular change, even though the disc may become very swollen. The retinal vessels will, however, bend sharply as they dip down from the swollen disc to the surrounding retina. The oedema may extend to the adjacent retina, producing greyish-white stria-

tions near the disc, and a white macular fan between fovea and disc may develop in some cases.

Papilloedema occurs in almost 80 per cent of all cases of brain tumour, but is particularly liable to occur in children with tumours of the cerebellum and fourth ventricle. It is uncommon in patients with pituitary tumours. An acute form of papilloedema with haemorrhage extending into the vitreous is characteristic of subarachnoid haemorrhage. A subdural haematoma may produce a similar clinical picture to that of a cerebral tumour. Papilloedema is uncommon in acute meningitis, but is more common in subacute and chronic meningitis. It may be the only physical sign in benign intracranial hypertension. Papilloedema occurring in malignant hypertension is accompanied by arterial changes characteristic of this condition and the haemorrhages and exudates extend far beyond the region of the disc.

**Optic neuritis.** Inflammatory, demyelinating, or vascular disease may attack any part of the optic nerve, producing an optic neuritis, the characteristic symptom of which is loss of vision, presenting as either a central scotoma or complete blindness. There is often pain on moving the eye, and the pupil on the affected side shows an ill-sustained contraction to a bright light. It is customary to divide optic neuritis into papillitis and retrobulbar neuritis.

(1) *Papillitis* is present when the disease process affects the optic nerve-head, producing hyperaemia and some swelling of the optic disc. It must not be confused with papilloedema, in spite of their similar ophthalmoscopic appearances. The two conditions can usually be distinguished by the gross visual loss that occurs with papillitis, as compared with the often minimal loss in papilloedema. In papillitis the swelling of the optic disc is usually slight, the distension of the retinal veins is less marked than in papilloedema, and there may be signs of inflammation (hazy vitreous, retinal exudates).

(2) *Retrobulbar neuritis* is present when the disease process affects that part of the optic nerve behind the eye. The same severe visual loss occurs as in papillitis, but the optic disc appears normal in the acute stage of the disease. Both papillitis and retrobulbar neuritis may be followed by optic atrophy.

**Optic atrophy.** In this condition the optic disc is paler than normal and may even be white (Plate XXI). Because of the wide variation in colour of the normal disc, a useful sign of optic

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atrophy is the reduction in small arterioles on the disc. In optic atrophy the number of small arterioles that cross the disc margin is reduced from the normal 10 to 7 or less. From the appearance of the disc it is customary, although not always very useful, to divide optic atrophy into primary and secondary types.

(1) *Primary optic atrophy*. In this type the disc is flat and white with clear-cut edges.

(2) *Secondary optic atrophy*. This follows swelling of the optic disc, due either to papilloedema or to papillitis. The disc is greyish-white in colour, it may be slightly swollen, and its edges are indistinct.

Optic atrophy may occur in a number of disorders, of which the following are a few.

(a) Interference with the blood supply to the optic nerve, as in occlusion of the central artery of the retina.

(b) Pressure on the nerve, whether in its intra-ocular, intra-orbital, intracanalicular, or intracranial portions.

(c) Following optic neuritis.

(d) Following trauma where the optic nerve or its blood supply is involved.

(e) In toxic conditions due to substances such as tobacco, alcohol, lead, etc.

(f) In certain congenital disorders, when it is frequently associated with other neurological signs.

(g) Following widespread chorio-retinal inflammation or degeneration.

**Opaque or medullated nerve fibres** usually present as one or more bright white patches radiating for a short distance from the optic disc (plate XXI). The patch has a characteristic feathered edge and retinal vessels may disappear for a short distance within it. This condition is a harmless and stationary congenital anomaly.

A **myopic crescent** is a ring of exposed white sclera, usually on the temporal side of the optic disc (plate XXI), but in some cases extending all round it. When marked it may be associated with other degenerative changes in the fundus, which, if they involve the macula, will result in reduction of central vision.

**Retinal haemorrhages** occur in a number of different conditions and are due to one or more of the following factors.

(1) Increased blood pressure within the retinal vessels, as in hypertension and chronic nephritis.

(2) Abnormalities in the walls of the retinal vessels, as in arteriosclerosis, diabetes mellitus, and occlusion of the central vein of the retina.

(3) Abnormalities in the circulating blood, as in severe anaemia, leukaemias, and bleeding diatheses.

(4) Sudden reduction in intra-ocular pressure, following a penetrating wound (surgical or traumatic) of the eye.

When superficial, within the nerve-fibre layer of the retina, the haemorrhages are elongated and 'flame-shaped', whereas when deep they are round blotches or spots. Subhyaloid haemorrhages, situated in front of the retina, are occasionally seen as very large round haemorrhages with a straight horizontal upper border; they sometimes occur in diabetic retinopathy and after a subarachnoid haemorrhage.

**Retinal arteriosclerosis** occurs either as an exaggeration of the general ageing process of the body or in association with hypertension. It is characterized by (1) broadening of the arterial light reflex, producing a 'copper-wire' or 'silver-wire' appearance; (2) tortuosity of the vessels; (3) nipping, indentation or deflection of the veins where they are crossed by the arteries; (4) white plaques on the arteries; (5) 'flame-shaped' haemorrhages and 'cotton-wool' exudates in the region of the macula.

**Hypertensive retinopathy** (Plate XXII) is characterized by a generalized narrowing of the retinal arteries, particularly in the young patient. In older patients these changes are masked by the accompanying arteriosclerosis. If the hypertension is severe, fullness of the retinal veins and 'flame-shaped' haemorrhages occur around the optic disc, and there is retinal oedema extending towards the macula, sometimes accompanied by a star-shaped collection of white exudates around the macula. In malignant hypertension papilloedema is also present. The retinopathy seen in some cases of acute and chronic nephritis is due to the associated hypertension.

**Diabetic retinopathy** (Plate XXII). The fundamental change in this condition is the formation of capillary micro-aneurysms, seen as tiny red spots around the macula. Micro-aneurysms are not seen in such abundance in any other condition. Retinal haemorrhages and exudates may occur; the haemorrhages are punctate or round, and the exudates have a waxy yellow-white appearance. The haemorrhages may extend into the vitreous and result in a glial proliferation

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called retinitis proliferans, which may result in blindness by covering the macula or by causing a retinal detachment. Patients with diabetic retinopathy often have associated arteriosclerotic or hypertensive changes in their fundi.

**Retinopathies in disorders of the haemopoietic system.** In severe anaemias the fundus may be paler than normal and a few small 'flame-shaped' haemorrhages and small woolly exudates may be present.

In polycythaemia the retinal vessels are dark, tortuous, and dilated. There may be oedema of the optic disc, and a few retinal haemorrhages may be observed.

In the leukaemias the retinal veins may be tortuous and dilated. In later stages of these diseases the arteries and veins may be yellowish in colour, and the fundus may have a generalized pallor. Retinal haemorrhages of various types may occur, the characteristic ones in leukaemia being round with a pale centre.

**Occlusion of the central artery of the retina.** The optic disc and surrounding retina are pale, and there is a cherry-red spot at the macula which contrasts with the milky pallor of the adjacent retina. The retinal arteries are narrow or even thread-like.

**Occlusion of the central vein of the retina.** There is intense swelling of the optic disc, with gross venous dilatation, and numerous retinal haemorrhages extend from the disc in all directions.

**Choroiditis.** In acute choroiditis there are one or more round or oval whitish patches in the fundus, lying deep to the retinal vessels. These patches have ill-defined edges and the vitreous may be hazy. When the acute phase subsides, flat white scars with pigment around their edges are left. The numerous causes of choroiditis include tuberculosis, syphilis (which may cause a disseminated choroiditis), and toxoplasmosis (which characteristically produces lesions at the maculae).

## II THE EAR

### Hearing

Hereditary deafness, usually recessive but sometimes dominant or sex-linked, is commonly the cause of hearing defects dating from birth. Many syndromes are described. Damage to hearing may occur from the effects of viruses on the acoustic apparatus of

the foetus by maternal rubella, or from teratogenic drugs, from prolonged anoxia or prematurity, or in any form of severe jaundice in the newborn. Methods of testing hearing in utero have been developed, but are beyond the scope of this book. The mother should be warned if her baby is 'at risk', and she can help by noting its reaction to sounds in its environment. The baby can be conditioned to interesting sounds (like the ringing of a tiny bell) just before feeds, and its responses noted if the feed is delayed. Unless the baby is able to hear the sounds of language, its neural mechanisms of speech will not develop normally.

Routine group audiometry tests are regularly conducted by school medical officers. If any abnormality is discovered, the external ear passage and the tympanic membrane of the child should be examined with an auriscope, in case there is a simple cause for the deafness (ear-wax, foreign body, infection, unsuspected congenital abnormality). It must be remembered that a child at school may lapse into inattention or become discouraged and irritable, if it has to 'listen' actively under strain for long periods, instead of hearing easily and naturally. A child may be partially deaf in only one ear, and have difficulty in focusing on sound, and yet his problem may escape casual notice.

The hearing of adults is often poor because of middle-ear infections in early childhood, which cause scarring and damage to middle ear conduction: virus infections damaging the cochlea; trauma from head injuries, or excessive exposure to noise, or sudden changes in air pressure (barotrauma); tumours; simple collections of ear-wax; otosclerosis, which produces a conduction deafness due to extra bone having been laid down round the oval window of the middle ear; or the hearing loss may simply be due to an upper respiratory infection with auditory (eustachian) tube obstruction.

The effect on hearing of noise in industry is becoming of more and more importance. Any background noise that necessitates raising the voice during normal conversation is said to be 'traumatic to hearing'. Continuous exposure to noise tends to produce permanent damage to perception of hearing, whereas intermittent exposure often allows recovery to take place. Permanent hearing loss from exposure to noise usually begins with cochlea damage at about 4000 cycles (high-tone deafness) and spreads to tones above and below that point.

*Tests of hearing will be found on p. 249.*

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### **Rinne Test (see p. 249)**

#### **Audiometry**

There are several interesting audiometric tests, but it will be sufficient to describe one. The audiometer is a simple instrument which allows the examiner to select on a dial a series of pure tones for testing air- and bone-conduction. Another dial allows the examiner to increase or decrease the intensity of each tone. The patient wears earphones, connected with the instrument, and indicates to the examiner when he begins to hear each separate tone. A graph or audiogram is then constructed which shows how much sounds of different frequencies must be intensified to be heard by the patient (Fig. 63). A series of such audiograms gives a good indication of the progress of the disease. The audiogram illustrates clearly the difference between conductive deafness (bone-conduction better than air-conduction) and perceptive deafness (air-conduction better than bone-conduction). Other information may be obtained from the shape of the curve, but this is beyond the scope of this book.

#### **Tests of Vestibular Function**

Disturbance of labyrinthine function, with intermittent attacks of rotational vertigo (objects rotating round the patient), nausea or vomiting, and nystagmus, are fairly common, and, because the vestibular apparatus and the cochlea share the same circulation, there is often associated deafness and tinnitus in the affected ear. These disturbances occur in Ménière's Disease; after head injuries, when the inner ear or vestibular pathways have been affected; when tumours in the posterior fossa exert pressure on the vestibular nerve; in vestibular (viral) neuritis; and when the blood circulation is interfered with in vertebral or basilar artery insufficiency.

The most satisfactory tests for vestibular function are caloric tests, because they are simple and inexpensive, and the results are remarkably constant.

The patient lies supine on a couch, with his head raised 30 degrees, to bring the lateral semicircular canal into a vertical plane so that it responds best to thermal stimulation. A stimulus, of cold water 7° C below body temperature (i.e. 30° C) and then, after an interval, one of warm water 7° C above body temperature (i.e. 44° C) is applied directly to the tympanic membrane and so indirectly to each labyrinth. Not less than 250 ml. water is run into

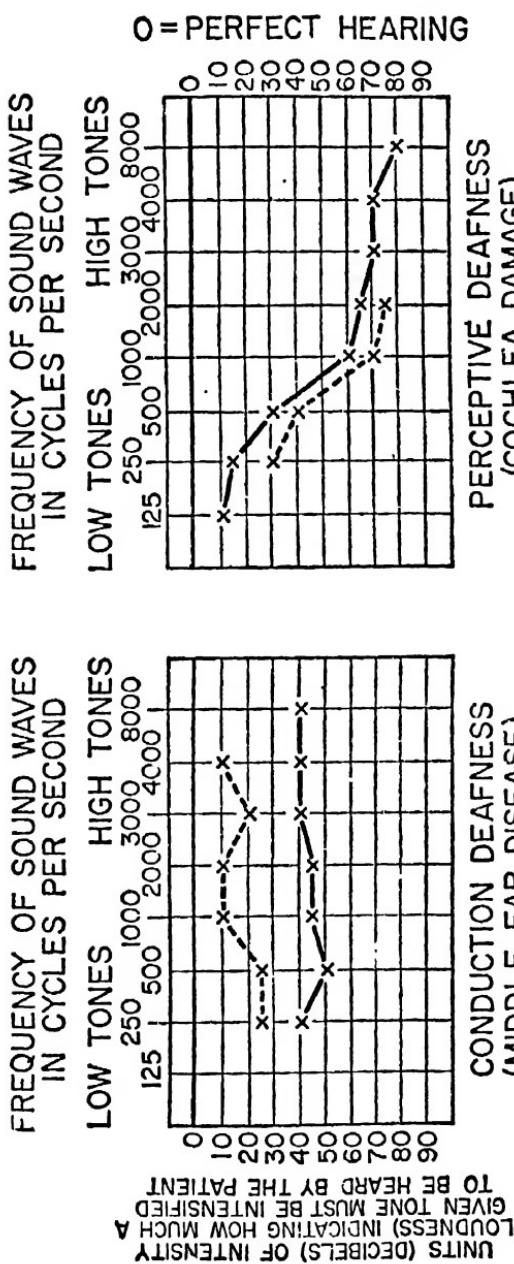


Fig. 63. Audiograms.

In conductive deafness (*left*) bone-conducted hearing is better than air-conducted, and low and high tones are equally affected. In perceptive deafness (*right*) air-conducted hearing is better than bone-conducted, and high tones are particularly affected.

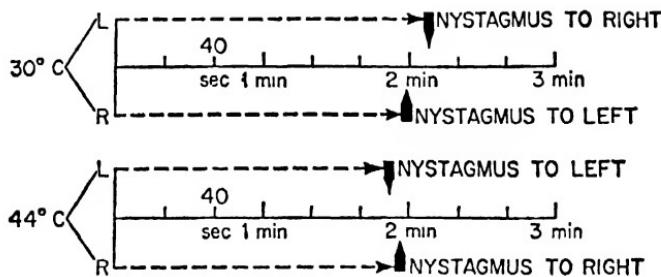


Fig. 64. Caloric tests.

Normal average response to stimuli applied separately to each tympanic membrane. The duration of after-nystagmus from the beginning of the stimulus is shown by the dotted line. The direction of after-nystagmus in terms of the quick component is also shown.

each external ear passage for 40 seconds continuously. In the normal person this produces horizontal nystagmus lasting approximately two minutes (Fig. 64). The caloric test is of great value in the diagnosis of organic lesions at all levels of the vestibular system. In peripheral lesions, the commonest abnormality found is canal paresis, and this is frequently demonstrated in Ménière's syndrome, vestibular neuritis, and acoustic neuroma.

#### Examination of the Ear

Note any unusual appearance of the external ear, and compare one ear with the other. Though there are many small variations in the shape of the 'normal' ear, occasionally genuine malformations occur, sometimes recurring in different generations of the same family, and sometimes associated with other deformities elsewhere, e.g. asymmetry of the face, cleft palate, spina bifida.

The commonest abnormality met with is probably the protruding ear or 'bat's ear'. Small accessory cartilaginous auricles may be detected, or a tiny fistula, because the development of the external ear is closely related to the branchial cleft between the first and second arches of the developing embryo. There may be only a rudimentary pinna, sometimes combined with the absence of any external auditory passage and this, of course, prevents normal hearing.

Movement of the pinna of the normal ear does not give rise to discomfort, nor does lying on it. It is best to ask the patient, or the infant's mother, whether the ear is painful to the touch, before the

examination begins. Always examine the 'good ear' first. Note any swelling or redness that might indicate infection in the skin (such as a small hidden boil), and note any bruising or cut in the skin due to recent trauma. Look at the opening of the external ear passage by gently drawing the pinna towards you with your finger and thumb. Note any desquamation or fissuring, or dermatitis aggravated by scratching with the finger, and glance at the hair and scalp for other evidence of dermatitis.

Make a careful note of any discharge from the opening of the ear passage. Thick pus may be coming from a recently ruptured boil; mucopus could be escaping from a perforation in the tympanic membrane, because the middle ear is lined by mucus-secreting membrane; blood or cerebro-spinal fluid may be escaping from a tear in the tympanic membrane from a fractured base of the skull, or blood alone may be coming from any recent tear in the skin of the ear passage.

It is often helpful to determine whether ear-pain or ear-discharge is of superficial origin or involves the middle ear. Middle-ear infections are nearly always secondary extensions of some pre-existing pharyngeal infection, and the infection reaches the middle ear via the eustachian tube, so that the history of any recent upper-respiratory infection is a help in diagnosis. The external cartilaginous ear may be moved freely without adding to the patient's discomfort, because tension from the middle-ear infection is confined to the tympanic membrane and the tympanic plexus of the middle ear, within the bones of the base of the skull, and movement of the outer ear does not disturb this. If, on the other hand, the ear-tenderness is due to a dermatitis, boil, or cut in the external cartilaginous ear-passage, any movement of the external ear will increase the patient's discomfort. Hearing is unaltered if the lesion is only a small boil or superficial cut, unless swelling of the skin itself occludes the air space of the lumen of the external ear passage. Hearing is always altered in middle-ear infections or haemorrhage. Of course, it is possible to have a middle ear infection and a dermatitis of the external ear passage at the same time, but more often the site of the lesion can be quite clearly defined.

Acute mastoiditis is not a common complication of ear infections today, thanks to early recognition and treatment of middle-ear infections with efficient antibiotics. Mastoiditis, which is a spread of infection from the middle ear cleft of the child to the surrounding bone, is an osteitis or osteomyelitis of the mastoid

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process. Pain or tenderness from mastoiditis is deep-seated, subperiosteal or periosteal, whereas the pain from a boil or from cellulitis tends to be superficial.

### **The Auriscope**

To examine the ear further, it is necessary to see the tympanic membrane. To do this, it is essential to illuminate the membrane, because the external auditory passage is only about 8 mm. wide in its greatest diameter and is about 3 cm. long. It is best to use a hand auriscope, which, like an ophthalmoscope, can be carried about and is powered by a small electric battery in its handle, and provides an adequate magnification, so that early changes in the tympanic membrane, indicating inflammation, may be speedily recognized.

A speculum of suitable size is fitted to the auriscope and the end of the speculum is then gently placed at the opening of the external ear passage, and the examiner looks upwards and forwards to view the tympanic membrane. For the right ear, the examiner holds the shaft of the auriscope in his right hand, like the shaft of a pen; with his other hand he gently pulls the pinna towards him, to straighten out the cartilaginous part of the passage. For the left ear, he holds the auriscope in his left hand. There are advantages in holding the shaft of the auriscope horizontally; for then, if the patient's shoulder is raised unexpectedly in defence, it will not 'jog' the instrument. The little fingers (of both hands) can rest on the patient's skull in front of or behind the ear under examination, and so prevent hurt to the patient should a child make any sudden or unexpected movement of its head.

The normal external auditory passage is very sensitive and is protected at its opening by hairs, sebaceous glands, and apocrine ceruminous glands, but there are no hairs or glands in the skin of the inner two-thirds, and here the skin lies directly upon the periosteum of the bone. The examiner, to his own advantage, should first practise a little with his auriscope, on the skin inside his own partly closed fist, and he will rapidly learn how close he should place his own eye to the lens of his examining auriscope, and will learn to adjust his own accommodation to the magnification. The focal length of the auriscope's lens is already fixed by the instrument-maker, and requires no adjustment, and the degree of magnification is standard for all general-purpose auriscopes. A gentle examiner will see more than one who is rough and inconsiderate, and an ear should be treated with as much respect as an eye.

Sometimes there is difficulty in seeing a tympanic membrane clearly. The commonest causes of difficulty are:

- (1) A fading electric battery, giving a poor light.
- (2) Collected 'wax' or cerumen, obscuring the view.
- (3) A dirty lens on the auriscope.
- (4) Faulty setting of the light bulb, so that the beam of light is not properly directed through the aperture of the speculum.
- (5) In new-born infants, the tympanic membrane is lying almost horizontally, and it will only become elevated to  $55^{\circ}$  as the child grows older; failure to remember this can cause difficulty in the small infant.

**The Tympanic Membrane.** The tympanic membrane (Fig. 65) is oval shaped, and is covered by a thin extension of skin from the walls of the external ear passage. This skin covers the fibrous layer of the membrane, which, on its inner surface, is lined by the moist mucous-membrane of the middle ear. The lower two-thirds of the membrane appear tense and have a faintly bluish tinge of colour, because of the shadow cast by the middle ear cavity behind it. The upper third of the tympanic membrane is pink in appearance, and

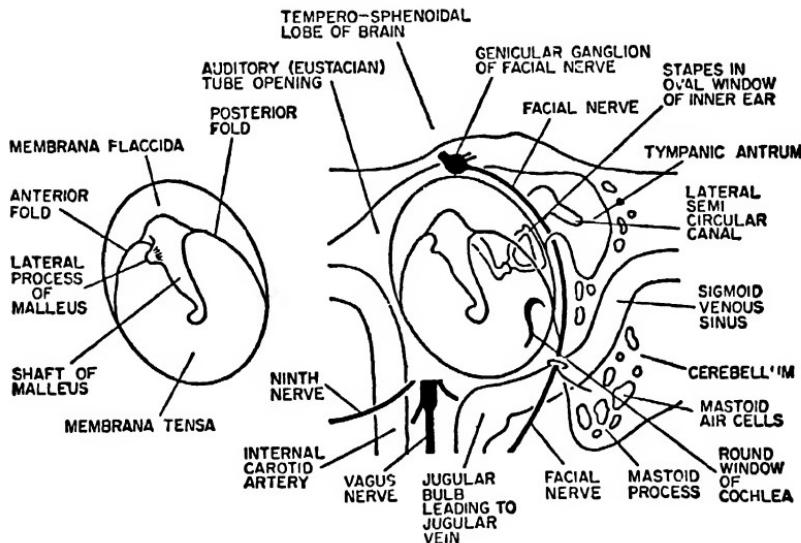


Fig. 65. The tympanic membrane and important anatomical structures around the middle ear.

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is more flaccid than the lower part. Near the centre is the shaft of the malleus, which is of a pale cream colour, and it is convenient for the examiner to identify the malleus first, in order to orientate himself. Sometimes necrotic middle-ear disease may have destroyed the shaft, and then the examiner should identify the lateral process, which often survives. The entire tympanic membrane may have been destroyed by disease, and the examiner will then identify some of the anatomical structures of the exposed middle ear (Fig. 65).

Any unusual redness or bulging of the tympanic membrane should be noted. Bulging or convexity of the tympanic membrane may indicate fluid, pus, or increased air pressure in the middle ear and retraction or concavity often indicate that the eustachian tube is obstructed. The malleus may appear severely retracted, if the ossicles have become partly fixed from old arthritic changes, for the small joints and ossicles of the middle ear suffer from these changes, just as do the larger joints of the body. If the auditory tube remains closed for long periods, serous fluid may collect in the middle ear, and then this may be seen through the tympanic membrane as a fluid-level which moves and forms bubbles which are easily distinguishable. It is worth remembering that in the new-born healthy infant, serous fluid normally fills the middle ear, and that this only disappears after air has entered the infant's middle ear cavity up the eustachian tube.

If a perforation of the tympanic membrane is present, it should be described according to its anatomical position (Fig. 65). There may be evidence of a recent tear in the tympanic membrane, with fresh or dried blood, after a head injury. It is always important to examine with an auriscope the ears of any patient, conscious or unconscious, who has recently suffered a head injury, and to make a note of what is seen. Cerebro-spinal fluid may be escaping through the ear from the meninges of the brain. Fluid pus, escaping from a perforation, may appear to pulsate, from arterial pulsation transmitted from the carotid (Fig. 65). The perforation may be old, resulting from failure to heal after some forgotten infection. In otosclerosis the tympanic membrane is intact, with limited conduction of sound and fixation of the ossicles. Osteomas are fairly frequently seen close to the tympanic membrane. They cause deafness. They appear as rounded hard swellings, and are common in swimmers. The irregular raised epithelium of a carcinoma is rare, fortunately, in the middle ear, but is often preceded by years of

chronic irritation and inflammation. A cholesteatoma is much more common. This is really a hyperkeratosis, with the formation of pearly-white epithelial tumours in the middle ear, and is often associated with chronic middle-ear disease. It is important, because it may not only destroy hearing but lead to intracranial complications. Glomus body tumours are rare, but appear as reddish swellings in the floor of the middle ear and are associated with paralysis of the ninth, tenth, and eleventh cranial nerves (Fig. 65).

### Radiological Examinations

Radiological examination of the ears is becoming more and more useful. Evidence of bone-disease, such as bone-erosion or sclerosis, may be present. A fracture may be clearly seen, but may be present but invisible by X-rays, because other bony structures may obscure it. Widening of the internal auditory meati may help in the diagnosis of acoustic nerve neuroma or posterior fossa tumours.

### Syringing the Ear

Syringing is used for the purpose of removing obstructions from the external ear-passage, mainly collections of cerumen, sebum, and keratin (collectively called wax), foreign bodies, or pus. The method is to direct a stream of water, at body temperature, along the sides of the ear passage so that it is deflected by the tympanic membrane and forces the obstruction out. There is an advantage in that the water may gradually soften the wax and make removal easier. If removal of wax by this method proves difficult, it should not be persisted with. It is sensible to ask the patient to soften the wax beforehand with olive oil or almond oil, dropped into the ear passage twice daily for a fortnight. Syringing with force is always liable to cause pain and damage. Even gentle syringing should never be used if a perforation in the tympanic membrane is suspected, and never if there has been recent ear-pain or evidence of a recent upper respiratory infection, because an inflamed ear drum, softened by oedema, is very easily damaged. No ear should ever be syringed blindly; repeated inspection with an auriscope is essential. The use of suction is now replacing syringing; but this method also requires gentleness and a good light. Wax solvents, such as benzine, may cause lasting damage to the ceruminous and sebaceous glands in the skin of the ear passage.

### Cleaning the Ear-passage

Thin twists of cottonwool (the twist must be of less diameter than the ear passage itself, to prevent a piston action), are wound on to the end of a thin disposable wooden applicator, so that the twist of cotton extends beyond the applicator by about six millimetres ( $\frac{1}{4}$  inch). The twists may be dipped in olive or almond oil to give added comfort, because the ear passage is very sensitive. The twists are gently introduced into the meatus under direct vision with a good light, and are gently rotated to remove pus or particles of desquamated skin. Contaminated swabs should be immediately dropped under disinfectant fluid to prevent cross-infection. Repeated inspection with an auriscope is essential.

Children sometimes put foreign bodies in their ear-passages. These almost always require removal under a general anaesthetic. Even an apparently simple and readily accessible foreign body becomes impacted very easily and can cause great pain and damage.

Stimulation of the peripheral branches of the vagus in the skin of the external ear passage may cause not only severe pain but laryngeal irritation, vomiting in children, and fainting in elderly people.

## III THE LARYNX

Examination of the larynx gives information about the adequacy of the respiratory airway and the causes of changes in the voice.

To perform laryngoscopy it is necessary to use a laryngeal mirror. This is a rounded mirror set at an angle at one end of a long thin metal handle (Fig. 66). The idea is to hold the back of the mirror lightly but firmly against the soft palate so that an image of the vocal cords will appear in the mirror.

The examiner seats himself facing the patient. He will have to sit very close to his patient, with his legs to the side nearest to the source of light. A standard lamp with a good bright beam is placed at the side of the patient, on a level with the patient's mouth, and its beam is directed at the examiner. For a right-handed examiner the light is placed at the patient's left. The examiner must wear a face mask to protect himself should the patient cough directly

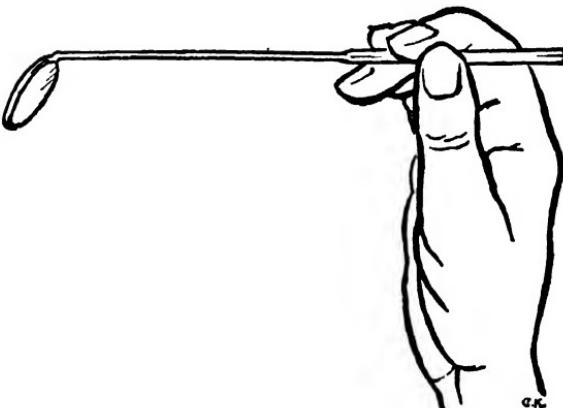


Fig. 66.—Holding the laryngeal mirror.

at him during the examination; most patients with serious lesions of the larynx are very helpful and co-operative, but anxious patients with slight diffuse inflammation of the larynx may be very sensitive, and cough easily. Next, the examiner places a head mirror on his forehead, over his right eye. A head-mirror is concave, and has a hole in the centre through which the examiner's right eye will inspect the image in the laryngeal mirror. The mirror is fastened to the head by a band, and can be adjusted easily because it is fitted with a ball-and-socket joint. The beam of light from the standard lamp is reflected on to this concave head-mirror, then on to the laryngeal mirror, and so down to the larynx. The examiner's eye looks directly down the centre of this reflected light-beam, and so the lighted image in the laryngeal mirror becomes clearly visible.

First the patient's mouth is inspected, and if artificial teeth are present, his dental plates should be removed. The laryngeal mirror is warmed by placing it in hot water, or holding it for a second over a spirit-flame. This is to prevent misting of the mirror when the patient exhales. The mirror is wiped clean, and tested to make sure it is not too hot by holding it firmly against the sensitive ventral surface of the examiner's wrist.

The patient is asked to lean a little forward, and to open his mouth and put out his tongue. With a small square of clean linen in the left hand, the examiner takes hold of the tip of the patient's tongue between his thumb and second finger. The linen (or

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gauze) square is necessary, or the fingers will slip on the moist tongue. The index finger of the same hand gently lifts the upper lip.

The light from the head-mirror is directed at the patient's mouth. The laryngeal mirror is held in the right hand (Fig. 66) and, very carefully in order to avoid touching the tongue, the small round mirror is placed deliberately and steadily against the soft palate near the base of the uvula. The shaft of light from the examiner's head mirror should now shine directly on to the laryngeal mirror, and the laryngeal mirror is gently adjusted until the vocal cords come into view.

Laryngoscopy requires no local analgesic in the great majority of examinations, but it does require the confidence of the patient in the examiner, and the examiner's confidence in himself. The examiner must train himself to make rapid and accurate observations.

To facilitate matters, the patient is asked to continue breathing throughout the examination. If the patient is concentrating on breathing he will not think of retching or coughing. If movement of the cords is required, he is asked to say 'E'.

The *larynx* should be inspected with great care (Fig. 67). The *epiglottis* lies in front, at the base of the tongue. Normally its upper curved edge is clearly seen, its colour is a pale yellow. Any abnormal position should be noted. The base of the *tongue*, and the *valleculae* are next examined. Both *piriform fossae* are inspected, and the presence of excessive froth or mucus round the *oesophageal opening* behind the larynx, or in either *piriform fossa*, should suggest upper

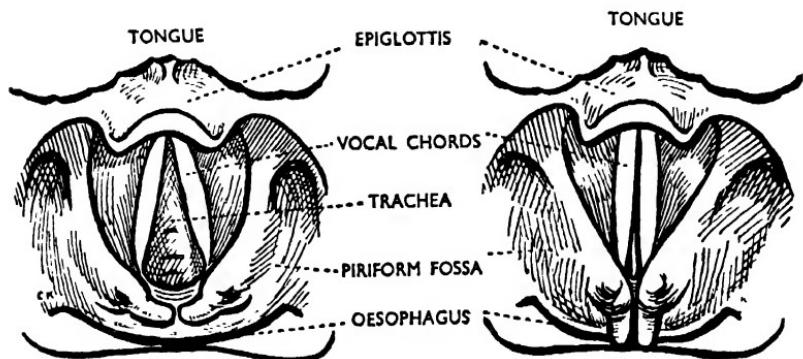


Fig. 67.—The larynx as seen in the laryngeal mirror.

oesophageal obstruction or paralysis. In upper oesophageal obstruction, the saliva and mucus from the mouth and upper respiratory tract cannot get away easily, and may overflow into the larynx, explaining the patient's irritating cough.

The *vocal cords*, or vocal folds as they are now sometimes called, are normally clear cut, and of a pale-yellow glistening appearance. The rest of the laryngeal mucosa is normally a moist pinkish-red colour. Between the vocal cords is seen the lumen of the *trachea*, and the outlines of the tracheal rings. The presence of any pus or mucus in the trachea, and any narrowing of the tracheal inlet, should be noted.

**Displacement** of the larynx suggests inflammation or new growth in the tissues closely adjacent to the larynx.

The *epithelium* should be inspected for any irregularity, ulceration, change of colour, or presence of oedema. Raised irregular epithelium suggests malignant changes. Ulceration with oedema makes one think of tuberculosis if there is evidence of pulmonary infection. Oedema alone indicates inflammation or trauma, or both. Diffuse redness of sudden onset suggests acute laryngitis.

The *movement* of the larynx should be noted. The movement of both sides of the larynx should be symmetrical. When normal movement is absent, the examiner must ask himself whether some local condition such as fixation due to malignant changes, or myopathy, is preventing normal movement, or whether some lesion along the course of the recurrent laryngeal nerves is responsible. If loss of movement is caused by some local change, there will usually be signs of swelling, or obstruction, or ulceration; or there may be a past history of poliomyelitis or diphtheria.

If the larynx looks quite normal, except for the paralysis, common sites along the courses of the recurrent laryngeal nerves worthy of clinical examination are: (1) the thyroid gland; (2) the apices of the lungs and structures related to the under surface of the aorta; and (3) the cervical oesophagus.

Absence of movement of one or both cords should be carefully noted. If one arytenoid lies a little in front of the other, this is often suggestive of unilateral paralysis, and may be helpful if the larynx is difficult to examine.

In *unilateral paralysis*, the examiner is struck by the overaction and compensation of the normal cord, which comes right across the middle line to meet the paralysed cord, if the patient endeavours to say the vowel 'E'. It is this remarkable compensation which

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explains the apparently normal voice in unilateral paralysis, and accounts for the tiredness of the voice towards the end of the day. There is usually no stridor, and no apparent obstruction to the airway. Compensation is rapid. Some very temporary difficulty with swallowing is experienced due to unilateral paralysis of the upper sphincter of the oesophagus (*crico-pharyngeus*) which is also supplied by the recurrent laryngeal nerve.

The extraordinary compensatory powers of the larynx are well seen in *bilateral paralysis* where, although the voice is undoubtedly weak, it is remarkably clear, those muscles not supplied by the recurrent nerves taking on added functions. Even when the complete larynx has had to be removed, because of malignant changes, and a permanent tracheotomy is the only respiratory passage, many patients develop an amazingly good voice. Folds in the pharyngeal walls are used for phonation, and the relaxed oesophagus makes an efficient air reservoir.

In unilateral paralysis, the patient does not complain of shortness of breath on exertion; whereas in bilateral paralysis there is great narrowing of the laryngeal airway, because the main abductors of the cords are paralysed, and the patient does complain of real shortness of breath on exertion. There is usually acute distress if both cords are paralysed suddenly, but with reassurance, and when adaptation is established, most patients complain of little difficulty in breathing when at rest, or during light movement. The *stridor* during sleep is very inconvenient for others. There is no stridor in unilateral paralysis. It must be remembered that recovery does take place more often than is usually realized, in both unilateral and bilateral paralysis of the recurrent laryngeal nerves.

It is very important to examine the larynx in any case of *hoarseness* that has persisted for two or three weeks. Intermittent attacks of hoarseness, or loss of voice, with a complete return to normal between attacks, should suggest simple laryngitis, or hysteria. If any growth is present on either vocal cord, the voice will never clear until the growth has been removed. Whenever the epithelium of the larynx is ulcerated, or irregular, an X-ray of the lung fields, and the Wasserman reaction, should be done before any question of biopsy is considered. If there is purulent sputum, this also should be examined.

When considering the advisability of *biopsy* as an aid to diagnosis, it must be remembered that interference with the edge of a growth (where the normal tissue-cells of the body are doing their

best to resist the encroachment of malignant cells) may cause rapid extension of the growth in the larynx. This is particularly important when a growth is small, and a complete cure is probable. In such cases, the best and safest diagnostic biopsy is often the removal of the complete tumour surrounded by an adequate margin of healthy tissue.

No laryngoscopy is complete without an examination of the neck. The position of the thyroid cartilage, and trachea, should be examined, and any displacement noted. Movement of the larynx on swallowing should be free, and any unusual lump, or enlarged nodes must be felt for.

#### IV THE NASAL PART OF THE PHARYNX

Examination of the nasal part of the pharynx is carried out in much the same way as that described above for laryngoscopy, and with the same precautions; only here the mirror is smaller, and its handle is bent to avoid touching the tongue. The tongue may be depressed with a spatula, and the small mirror is placed just behind the soft palate facing upwards. The examination requires considerable skill and confidence, and, as with laryngoscopy, some patients are easy to examine, and some very difficult indeed.

The *posterior nares*, lying to either side of the sharply defined vomer, come into view in the mirror, and the posterior ends of the inferior and middle conchae can be seen. To either side lie the openings of the *eustachian tubes*. The presence of any tumour or pus, or change in the normally pink moist epithelium should be noted.

#### V THE NOSE

The outside of the nose must be inspected first. Old scars may indicate past trauma. Recent cuts or bruising should be noted. Redness or swelling may be caused by inflammation. Displacement of the *nasal bones*, to right or left, or depression of the bridge suggests trauma. If the bones are firm and painless, the injury will not have been of recent origin. Collapse of the bony or cartilaginous *bridge* may have followed necrosis. With a finger and thumb on the outside of each nostril, the anterior part of the cartilaginous *septum* can be palpated, and any gross displacement or thickening of the *septum* detected.

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The skin round the *nostrils* must be inspected for fissuring or redness. The presence of discharge, whether blood-stained, or mucopurulent, or watery, must be noted, and whether the discharge is confined to one nostril only, or comes from both.

To inspect the *vestibule* of the nose, and the nasal passages, a head mirror is needed. There are several advantages in using a concave head mirror for examinations of the nose, naso-pharynx and larynx. The examiner can look right down the centre of the beam of light which his head-mirror reflects into these narrow and darkened passages. Both hands of the examiner are free, and his eyes are shaded. Other forms of light such as torches or head-lamps or lighted speculae can be tried, but they are not as satisfactory as the perforated head-mirror with reflected light.

The vestibule of a small child's nose, and the openings of its nasal passages, can be inspected by tilting the nose upwards with a finger.

To examine an adult's nose properly a *nasal speculum* is needed. The speculum is held in the left hand (Fig. 68) and the light beam

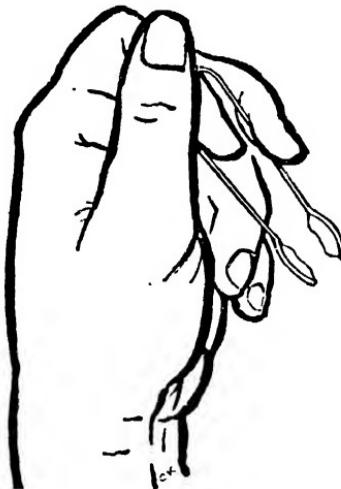


Fig. 68.—Holding the nasal speculum.

reflected from the head mirror is directed on to the patient's nostril. The speculum is closed, and gently introduced into the nostril. The spring is allowed to open a little, but never to its fullest extent. Gentleness is all important.

The skin of the vestibule extends inwards for about half an inch,

and it is well supplied with protective hairs, particularly in males. The skin of the nasal vestibule is heir to all the common skin diseases. There is a clear line where the pale skin meets the pink moist mucous membrane of the nasal passages. Medially lies the *nasal septum*, which is commonly displaced by trauma. A haematoma of the nasal septum causes a swelling which is soft to the touch, unlike the normal septum, which is firm. Note the presence of any ulceration or perforation of the septum, or the presence of any bleeding point. Laterally, is seen the *inferior nasal concha*. This should be moist and pink, and as its epithelium can become pale and greatly thickened in hay fever and asthma, comparison should be made with the normal. Above the inferior concha can be seen the anterior part of the *middle concha*. The presence of pus coming from under the middle concha strongly suggests purulent sinusitis; for most of the accessory nasal sinuses open under this concha. Nasal polypi appear as moist greyish swellings occluding the air-passages. They are easily movable and painless unlike the conchae, which are fixed and tender. Hyperaemia of the mucosa and the presence of muco-pus in the nasal passages, probably indicates infection. A persisting one-sided blood-stained nasal discharge may indicate the presence of a foreign body, or malignancy if present in an older person. The presence of any raised irregular or bleeding epithelium should be noted.

The *nasal passages* form part of the upper respiratory tract, and should never be examined without reference to the lower respiratory tract. All air reaching the lungs has to pass through the nasal passages to be filtered, warmed and moistened, and any infection or obstruction in these passages may ultimately have some effect on the health and function of the lungs and bronchi. The presence of nasal obstruction must be noted. In children, mouth-breathing due to nasal obstruction gives rise to a change in the pattern of facial expression, which is indicated by the open mouth and sagging of the facial muscles. The lips and mouth are dry.

Severe nasal obstruction may prevent air reaching the olfactory area, and so cause loss of the sense of smell.

## VI THE NASAL SINUSES

To examine the *maxillary sinuses*:

(1) Palpate the bony walls of the sinus, giving particular attention to the bone under the eye. Compare the outline on the two

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sides. Any tenderness, swelling, expansion or depression of bone must be observed. Malignant changes in the epithelium of the maxillary sinus may remain undetected for many months. The palate and alveoli must also be inspected, and palpated from inside the mouth.

(2) Examine the nasal passages, to detect any evidence of pus or polypi appearing from any of the normal openings of the sinuses.

(3) Transilluminate the sinuses. This is done by placing a strong light in the centre of the hard palate. Any upper denture should be removed. The patient closes the lips round the handle of the transilluminating lamp. The examination should be performed in a dark room, or with curtains drawn round the bed. Light from inside the mouth shines through the hollow sinuses, producing a light in the bone under each eye, and a reddish reflection from the retinae.

Comparison between the two sides is made. Pus in the maxillary sinus throws a dark shadow. Patients will differ greatly in the thickness of their maxillary bones. Some transilluminate clearly, others with difficulty. Transillumination is a useful clinical aid, because if the sinuses are perfectly clear on transillumination, an X-ray may be unnecessary.

If any abnormal shadow is found, an X-ray of the sinuses will give additional information, and will help to confirm the clinical examination.

To transilluminate the *frontal sinuses*, the light is placed under the frontal ridge of the orbit. The *ethmoid* and *sphenoid* sinuses are best examined by X-ray, combined with clinical inspection of the nasal passages.

# 11 LOCOMOTOR SYSTEM

The locomotor system includes the muscles, bones and joints. The examination of the muscles is most conveniently considered along with that of the nervous system (Chap. 9). There remain for consideration the bones and joints.

## I THE BONES

In examining the *long bones* of the limbs, look for any alterations in shape or outline, for localized swellings in the bone, for signs of fracture and for evidence of undue tenderness. Alteration in the shape of the bones occurs particularly in rickets (Plate X). In osteitis deformans the bones are both deformed and enlarged (Plate XI). Localized swellings are mostly due to surgical conditions. Spontaneous fractures may occasionally assist in the diagnosis of carcinoma, generalized osteitis fibrosa (hyperparathyroidism), osteogenesis imperfecta or multiple myeloma. Undue tenderness of the bones, apart from surgical conditions, is found in generalized osteitis fibrosa, myelomatosis, occasionally in carcinomatosis of bones, and very rarely in leukaemia.

The *vertebral column and skull* demand special attention. Note the presence of any local projections or angular deformity of the vertebral spines, and state which vertebrae are involved and at what level the projection is most prominent. Landmarks are C.7 (vertebra prominens) and the last rib, articulating with the 12th thoracic vertebra. In many cases, however, the last rib cannot be distinctly felt and is therefore rather untrustworthy as a guide.

Note also any curvature of the spinal column as a whole, or of part of it, distinguishing carefully such general curvature from the local projections above referred to.

The curvature may be in an anterior, posterior or lateral direction. Anterior curvature (extension deformity) is termed *lordosis*,

and is commonest in the lumbar region. General posterior curvature (flexion deformity) is spoken of as *kyphosis*. It occurs most typically in the thoracic region in old persons, and must be distinguished from the localized angular deformity of Pott's disease. Lateral curvature is termed *scoliosis*, and may be towards either the right or the left side. It is always accompanied by a rotation of the bodies of the vertebrae in such a way that the spines come to point towards the concavity of the curve, i.e. the curvature is greater than it appears from inspection of the posterior spinous processes. Kyphosis and scoliosis may often be combined.

Ask the patient to stoop down; notice the degree of mobility of the vertebral column and the occurrence of any pain during stooping, noting the exact site of the latter if present. Then pass the hand down the vertebral column, and observe whether any tender spots can be discovered. To elicit more deep-seated tenderness of the vertebrae, it may be necessary to 'punch' the spines gently with the fist from above downwards, observing the point at which the patient complains of pain, and verifying the observation by repeating the process from below upwards.

In studying the *skull*, note its *shape*.

Is it of the dolichocephalic (long-headed) or the brachycephalic (bullet-headed) type? Certain well-recognized types of abnormal skull are met with. In *acromegaly* the supra-orbital ridges and bones of the face, particularly the lower jaw, are enlarged, so that in relation to them the calvarium may appear small (Plate XI). In *achondroplasia* the skull, though of approximately normal size, appears very large in contrast to the generally small stature. In addition, the bridge of the nose is greatly depressed and the nostrils tend to point directly forwards. In *osteitis deformans* (Paget's disease), in addition to the widening and bowing of the long bones, the skull is often greatly enlarged, particularly in its transverse diameter, so that it appears to bulge above the ears (Plate XI).

In *hydrocephalus* the skull tends to assume a globular form (Plate XI). The forehead is overhanging and the eyes are pushed down so that the upper part of the sclerotic is exposed. The lateral aspects of the skull (above the ears) project outwards. If the patient is a child, as is usually the case, the fontanelle is wide and bulging, and often fluctuates very distinctly. The sutures may be opened up, and imperfectly ossified areas (*craniotabes*) may be detected in the bones. In *rickets* the skull tends to be square or oblong and box-shaped. The frontal and parietal bones

often show central thickening ('bossing'). The forehead, however, does not overhang, nor are the eyes depressed, and although the fontanelle is usually widely open, it does not bulge as it does in hydrocephalus, nor are the sutures opened up. In *congenital syphilis* the forehead is vertical, the frontal eminences are often exaggerated, and the bridge of the nose is depressed.

## II THE JOINTS

These should be examined by inspection and palpation, and by tests for their range of movement. It is important to proceed in a routine manner—e.g. the jaw, cervical spine, shoulder girdle and upper limb, thoracic and lumbar spine, pelvis and lower limb, so that inconspicuous but important joints like the temporo-mandibular, sterno-clavicular and sacro-iliac will not be overlooked; and always to compare the corresponding joints on the two sides of the body.

On inspection and palpation look for enlargement or irregularity of the joint; for redness, tenderness and heat, and note whether the overlying skin is dry or moist. Tenderness may be recorded in four grades, depending upon the patient's reaction to firm pressure of the joint between finger and thumb. Grade 1 tenderness—the patient says the joint is tender. Grade 2—the patient winces. Grade 3—the patient winces and withdraws the affected part. Grade 4—the patient will not allow the joint to be touched. Grade 4 tenderness only occurs in gout, rheumatic fever or suppurative arthritis. In gout the skin overlying the affected joint is dry, whereas in suppurative arthritis or rheumatic fever it is moist.

Look also for bony outgrowths, such as Heberden's nodes on the fingers in some cases of osteoarthritis, for rheumatoid nodules, for gouty tophi and for atrophy of muscles in the region of the joint. If the joint is enlarged, determine whether the enlargement is due to effusion into the joint space, when it normally has a characteristic shape and fluctuation can often be elicited; to thickening of the periarticular tissues, such as occur in the rheumatoid type of arthritis; to enlargement of the ends of the bones, such as occur in pulmonary osteo-arthropathy; or to complete disorganization of the joint with absence of pain sense such as occurs in neuropathic (Charcot's) joints. If tenderness is present localize it as accurately as possible and determine particularly whether it

arises in the joint or in neighbouring structures, e.g. in the supraspinatus tendon rather than in the shoulder joint. Feel the joint with one hand, while it is moved passively with the other. A grating or creaking sensation known as *crepitus* may be felt. This often indicates osteo-arthritis, but not invariably so, for crepitus is commonly felt in the shoulder joints of older persons, whereas osteo-arthritis of these joints is rare.

In examining joints for the range of movement an estimate of the degree of limitation present based on previous experience or on comparison with the normal side may often be sufficient, but for accurate description the actual range of movement should be measured with a protractor or goniometer. In either case it is important that the student should be able to describe the movements of the various joints correctly. In carrying out the examination, both active and passive movement should be tested where possible, and the greatest possible gentleness must be exercised, particularly in the case of painful joints. Limitation of movement in a joint may be due to pain, muscle spasm, contracture, inflammation or thickening of the capsules or periarticular structures, effusion into the joint space, bony or cartilaginous overgrowths, bony ankylosis, or to painful conditions quite unconnected with the joint.

In describing the range of movement of joints the scheme shown in the following pages (modified by permission of the authors and publishers, from Cave, E. F., and Sumner, M. R., *J. Bone Jt. Surg.*, 1936, XVIII, 455) will be found useful. All motion should be measured in degrees from a neutral position or zero which must be defined whenever possible.

*Spine.*—Neutral position is normal upright position for patient, but cannot be further defined. Test:

- (1) Forward bending.
- (2) Extension.
- (3) Lateral bending.
- (4) Rotation with pelvis fixed, comparing angle made between axis of shoulders and that of the pelvis.

These movements cannot conveniently be measured, but should be compared with the probable normal for the patient's age.

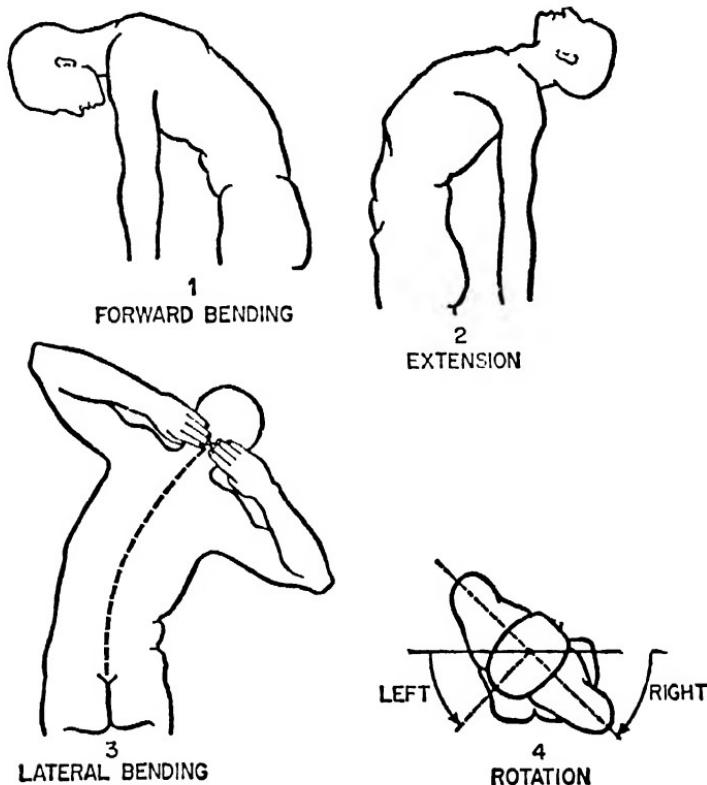


Fig. 69.

*Neck.*—Neutral position is that with head erect and chin drawn in. Test:

- (1) Rotation right and left.
- (2) Flexion.
- (3) Extension.
- (4) Lateral bending.

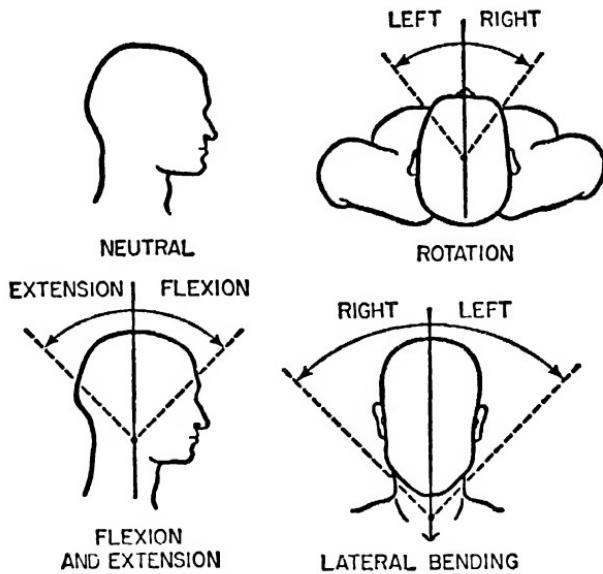


Fig. 70.

*Shoulder.*—Neutral position is arm to side, elbow flexed to 90 degrees with forearm pointing forwards. Test:

- (1) Flexion.
- (2) Extension.
- (3) Abduction.
- (4) Rotation in abduction.
- (5) Rotation in neutral.
- (6) Elevation (this is shoulder girdle motion as compared with 1-5 which are humeroscapular motion).

## MOVEMENTS OF THE SHOULDER

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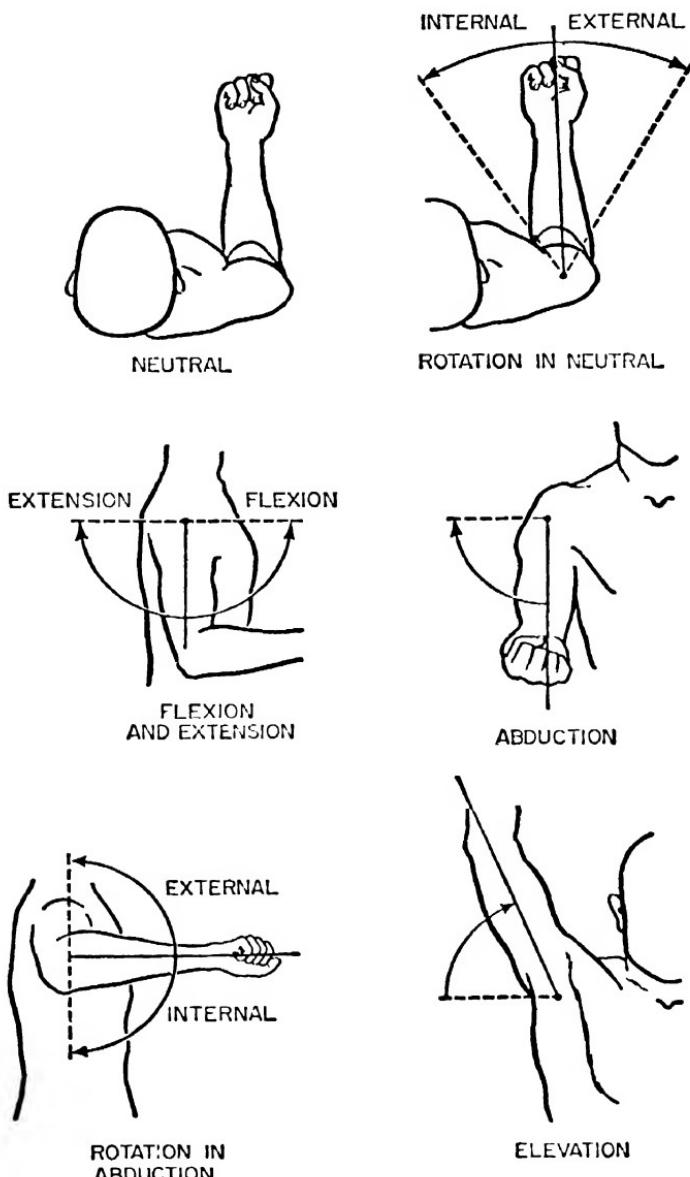


Fig. 71.

*Elbow.*—Neutral position is with forearm in extension. Test:

- (1) Flexion.
- (2) Hyperextension.
- (3) Supination { from neutral which is mid-point between supination and pronation, with elbow fixed
- (4) Pronation      at side in 90° of flexion.

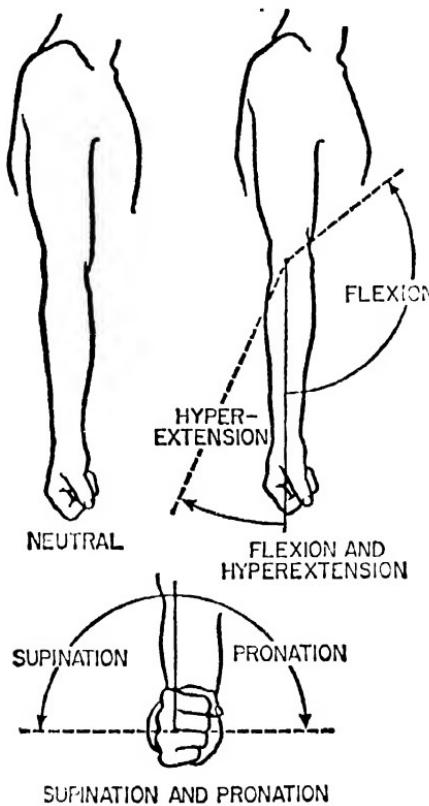


Fig. 72.

*Wrist.*—Neutral position is with hand in line with forearm and palm down. Test:

- (1) Dorsiflexion (extension).
- (2) Palmar flexion.
- (3) Ulnar deviation.
- (4) Radial deviation.

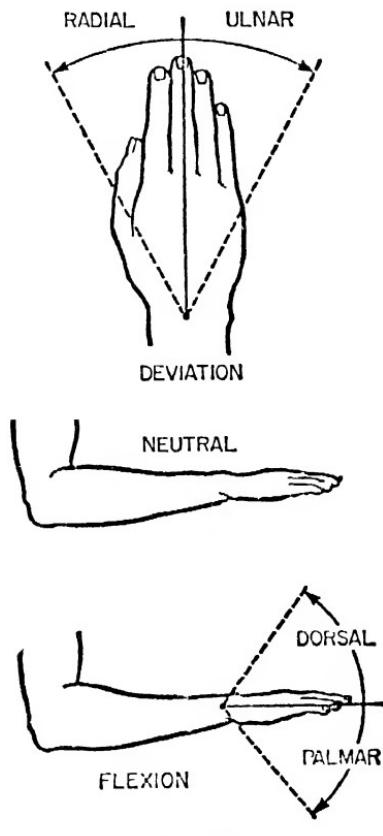


Fig. 73.

*Fingers.*—Neutral position is with fingers in extension. Test:

- (1) Flexion at metacarpo-phalangeal and inter-phalangeal joints.

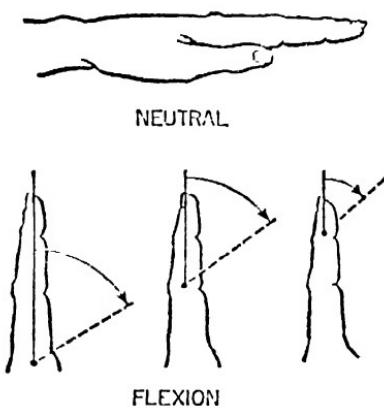


Fig. 74.

*Thumb.*—Neutral position is with thumb alongside forefinger and extended. Test:

- (1) Extension.
- (2) Flexion—measured as for the fingers.
- (3) Opposition.
- (4) Abduction (not illustrated) is movement at right angles to plane of palm.

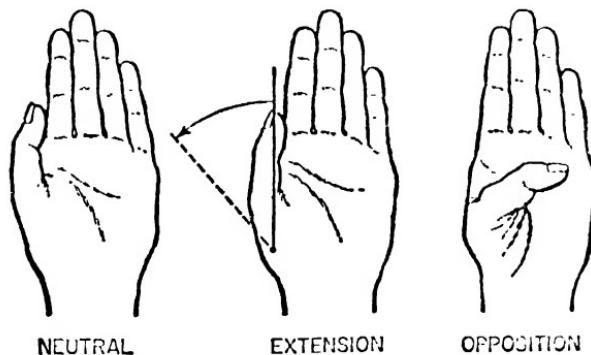


Fig. 75.

*Hip.*—Neutral position is with hip in extension, patella pointing forwards. Test:

- (1) Flexion, measured with knee bent. Opposite thigh must remain in neutral position.
- (2) Abduction, measured from a line which forms an angle of 90° with a line joining the anterior superior spines.
- (3) Adduction, measured in the same manner.
- (4) Rotation in flexion.
- (5) Rotation in extension.

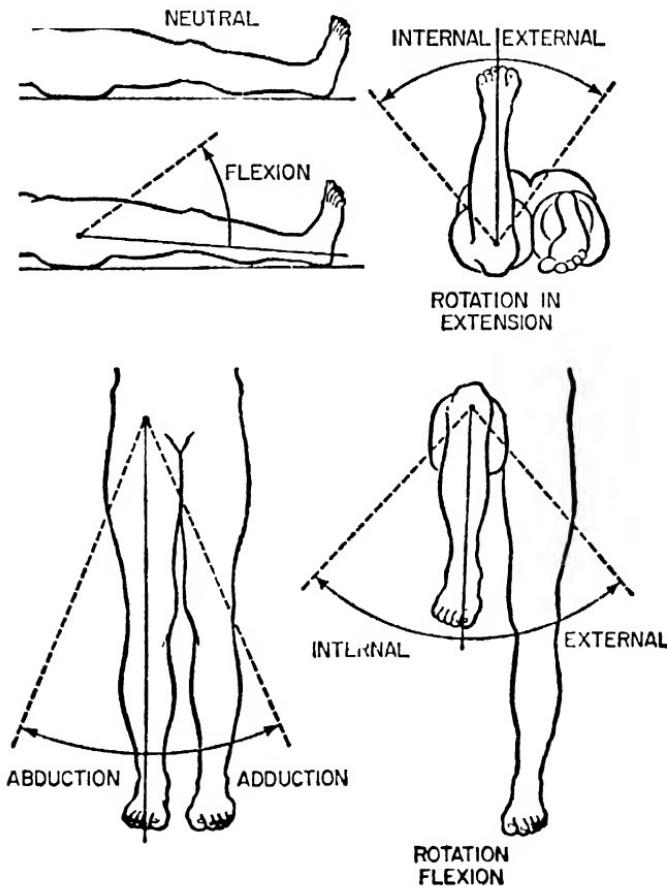


Fig. 76.

## Additional examinations of the hip joint:

## (1) Testing for extension.

Attempt to extend the hip with the patient lying in the lateral position on the opposite side. Extension should normally be at least  $20^{\circ}$  from the neutral position. This movement is lost early in hip joint disease, and the loss is associated with a flexion deformity and a 'waddling' gait.

## (2) Measurement of 'true' and 'apparent' shortening.

If the length of the legs is measured from the anterior superior iliac spine to the medial malleolus on the same side, any difference is referred to as 'true' shortening, and almost invariably indicates disease of the hip joint or the neck of the femur on the shorter side. If the length of the legs is measured from the umbilicus to the medial malleoli, any difference is referred to as 'apparent' shortening, and may be due either to disease as mentioned above or to tilting of the pelvis, usually due to an adduction deformity of the hip.

**Knee.**—Neutral position is complete extension. Test:

- (1) Flexion.
- (2) Hyperextension.

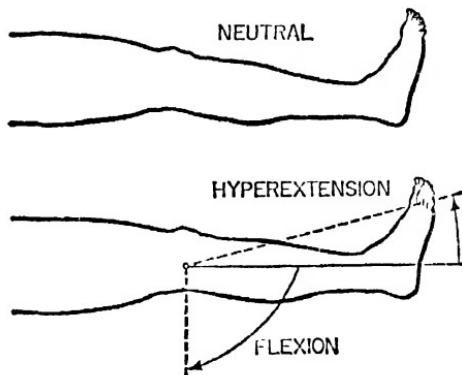
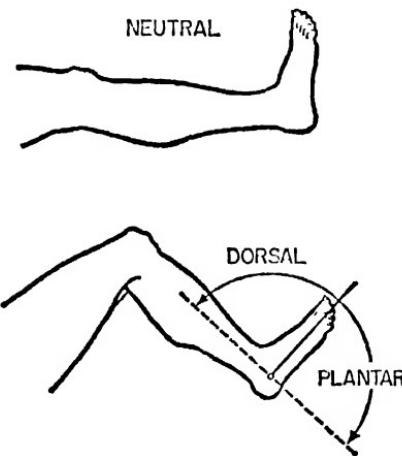


Fig. 77.

**Ankle.**—Neutral position is with the outer border of foot at angle of  $90^{\circ}$  with the leg, and midway between inversion and eversion.

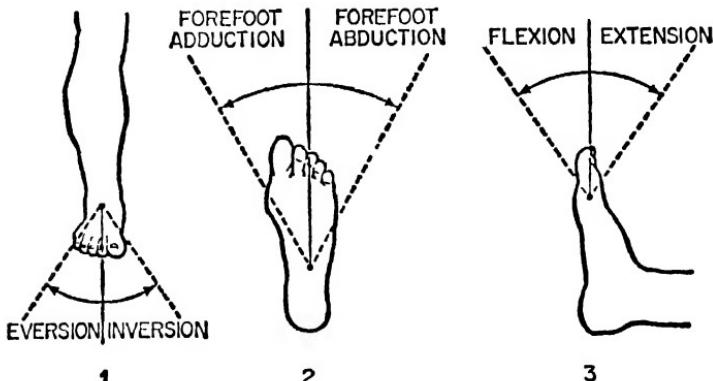
**Test:**

- (1) Dorsiflexion, with foot in inversion. Test with knee in flexion and extension to exclude tight calf muscles.
- (2) Plantar flexion.

**Fig. 78.**

*Foot.*—Neutral position cannot be defined. Test:

- (1) Inversion and eversion at sub-astragalar joints.
- (2) Forefoot adduction and abduction at mid-tarsal joints, with os calcis held in neutral position.
- (3) Flexion at metatarso-phalangeal and inter-phalangeal joints.

**Fig. 79.**

### III THE GAIT

The character of a patient's gait is often important in diagnosis. It is especially important in cases of neurological disease.

In studying the gait, it is well to have the legs fully exposed. The patient should therefore either be unclothed or wear a small triangle or bathing dress. The feet should be bare. The patient is told to walk away from the observer, to turn round at a given point, and then to come towards him again.

In studying the gait, the *points to be noted* are: (1) Can the patient walk at all? If he can—(2) Does he pursue a straight line, or does he tend to deviate to one side or the other, or to both alternately? To bring out this point, ask him to walk along a straight line, e.g. a crack in the floor. (3) Does he tend to fall, and, if so, in what direction? The next point to be decided is whether the gait conforms to any of the well-recognized abnormal types. Before deciding this, be quite sure that the peculiarity in the patient's gait is not due to some surgical cause or to local disease of a joint, e.g. osteo-arthritis of the hip. A previous examination of the bones and joints will eliminate such sources of error.

The three chief types of abnormal gait due to neurological affections are:

1. The spastic.
2. The ataxic.
3. The reeling.

It is usually sufficient to state that the gait belongs to one or other of these types, or to two or more combined. The chief peculiarities of each variety are as follows:

1. The *spastic* may be described as a 'sticky' gait. The patient has difficulty in bending his knees, and drags his feet along as if they were glued to the floor, the toes scraping the ground at each step. The foot is raised from the ground by tilting the pelvis, and the leg is then swung forwards so that the foot tends to describe an arc.

This gait is seen most characteristically in patients with pyramidal lesions. The *hemiplegic gait* is essentially a spastic gait in which only one leg is affected.

2. The gait in *sensory ataxia* may be described as 'stamping'. The patient raises his feet very suddenly, often abnormally high, and then jerks them forward, bringing them to the ground again

with a stamp, and often heel first. He may be fairly steady, if he watches the ground, as he uses his eyes in place of his sense of position, but becomes severely ataxic when his eyes are closed. This gait is best seen in cases of tabes dorsalis, and other signs of loss of postural sensibility will be present.

3. The gait of *cerebellar incoordination* may be described as a 'drunken' or 'reeling' gait and requires no further description. Patients with this gait walk 'on a broad base', the feet being planted widely apart. The ataxia is equally severe whether the eyes are open or closed.

This gait occurs in disease of the cerebellum or cerebellar tracts, and other signs of motor ataxia will be present. A similar reeling gait may be seen in patients under the influence of alcohol or narcotic drugs.

Some rarer varieties of abnormal gait are:

The *festinant gait*. This is met with in typical cases of Parkinsonism. The patient is bent forwards, and advances with rapid short shuffling steps, so that 'he looks as if he were trying to catch up with his centre of gravity', and the arms do not swing. In some cases, if he is suddenly pulled backwards, he begins to walk backwards, and is unable to stop himself, though he is leaning forwards all the time. This peculiar phenomenon is spoken of as *retropulsion*.

The *waddling* or oscillating gait is like the gait of a duck. The body is usually tilted backwards, there being a degree of lordosis present; the feet are planted rather widely apart; and the body sways more or less from side to side as each step is taken. The heels and the toes tend to be brought down simultaneously. The chief peculiarities of this gait are due to a difficulty in maintaining the centre of gravity of the body owing to weakness of the muscles of the back. It is met with in pseudo-hypertrophic muscular dystrophy and in congenital dislocation of the hips.

The *high-stepping* or prancing gait is a device adopted by the patient to avoid tripping from his toes catching the ground. It is, therefore, met with in cases where the toes tend to droop from weakness of the extensor muscles, e.g. in peripheral neuritis affecting the anterior tibial nerve. The name sufficiently describes its characters.

## **12 CLINICAL ASSESSMENT OF METABOLIC DISTURBANCE**

Disturbance of the metabolic state of the body produces disease with symptoms and signs as important in every respect as are those associated with structurally or histologically recognizable diseases of particular tissues. The severity of the disturbance of body function frequently relates as much to the rapidity of onset of the disturbance of metabolism as to its severity, as in many instances gradual changes in one aspect of metabolism—particularly in electrolyte disorders—are offset in part by other changes which protect the cells or the body as a whole. When the body is rapidly depleted of a particular constituent, there may be insufficient time for such compensation to occur, hence more serious illness results.

In considering the clinical assessment of disorders of fluid and electrolyte or mineral content in the body, attention must be paid in taking the history both to circumstances which may have led to loss or gain of particular body-constituents, and to the symptoms which result from a particular metabolic disorder. The concept of *balance* of intake and loss of fluid, minerals, and electrolytes is important, and in many instances a rough assessment of this balance must be made from the history, relating to food, liquids, vomiting, urinary output and faecal loss. After assessment of the previous balance, careful observation of these factors under hospital conditions may be necessary, with accurate estimation of both intake and loss for every 24-hour period. In assessing fluid-balance, allowance must also be made for *insensible loss* due to evaporation of water from skin and lungs, which in 24 hours amounts to 500–1200 ml. according to temperature and humidity. Such observations demand the cooperation of the patient, nurse and doctor, but if carefully conducted provide invaluable information. Thus, if deficiency of an electrolyte results from lack of intake, failure of absorption, or loss from the gastro-intestinal tract, the kidneys will then ordinarily excrete less of that electrolyte in an endeavour to maintain the

electrolyte environment in its normal state; and if the electrolyte is replaced either by mouth or the intravenous route, the kidneys will continue to conserve that electrolyte until the body is replete. Exceptions to this rule exist when renal function is not normal, or when the disease is one primarily of the regulating mechanism (as with an endocrine tumour, such as a parathyroid or adrenocortical adenoma), and here balance-studies may be essential to establish the diagnosis, by demonstrating that physiological balance no longer pertains in respect of the particular mineral or electrolyte concerned.

Full balance-studies are, of course, not often required, but none the less the mental discipline of thinking in terms of profit and loss, of intake and excretion, is essential to the understanding of most metabolic problems.

### ANATOMY OF BODY FLUIDS AND ELECTROLYTES

In a normal adult weighing 65 kg., water accounts for 40 kg. of the body-weight. This is subdivided into intracellular water of approximately 25 litres, and extracellular fluid of around 15 litres. The extracellular fluid is made up of some 3 litres of plasma and 12 litres of interstitial fluid. These figures are used for the estimation of the total deficit of electrolytes or water in certain circumstances.

The main cationic constituents of body electrolytes are sodium, potassium, calcium and magnesium. The distribution of these ions within the various fluid compartments varies very greatly, and an understanding of this distribution is essential to the interpretation of the significance of alterations in plasma concentration of the electrolytes, the only site in the body where content of the electrolytes can be readily measured.

It will be seen from the table that the main intracellular cation is potassium, the extracellular potassium representing only a small portion of the body-content. The reverse is true of sodium. Much of the body calcium and body magnesium is contained in the bones, and is not in soluble ionic form, and in plasma much of the calcium present is bound in a complex with protein and organic anions, and thus only a portion is represented as fluid cation.

## DISTRIBUTION OF CATIONS IN BODY FLUIDS

	<i>Intracellular</i> Concentration <i>mEq/l</i>	<i>Content</i> <i>mEq</i>	<i>Extracellular</i> Concentration <i>mEq/l</i>	<i>Content</i> <i>mEq</i>
Sodium	10	240	145	2,320
Potassium	150	3600	5	80
Calcium	2	48	2	32
Magnesium	15	360	2	32

## FLUID, SODIUM AND POTASSIUM DISTURBANCE

**Water depletion**

This is uncommon as an isolated disorder, being in most instances associated with salt depletion (see below). However, it may occur through water deprivation, or through uncontrolled excretion as in diabetes insipidus. The body may lose two litres of water, and the only symptom will be thirst and some weakness. However, with the loss of four litres there is profound weakness and disorientation. The important signs are those of dry mouth and skin, soft eyeballs, and rapid pulse, and except in diabetes insipidus, the urine specific gravity is greatly increased. (Measurement of urine specific gravity is simply done, using a hydrometer, and is of the utmost importance.) In the plasma, the serum sodium-concentration is above 145 mEq/l, and may rise to as high as 155 mEq/l. The concentration of other electrolytes is increased in proportion.

**Water excess**

Water intoxication not infrequently occurs as a result of incorrect treatment of some other fluid and electrolyte disorder. Also it may complicate states of disordered excretion of water in acute renal failure or endocrine disorders.

Symptoms of water intoxication are headache, cramps, drowsiness, muscular twitching and fits. The mucosae are moist, but pitting oedema is not commonly found. The urine is dilute with a low specific gravity, unless renal failure or inappropriate secretion of anti-diuretic hormone has occurred (a rare disorder sometimes

found with bronchial carcinoma, hypothalamic tumours, and occasionally in cirrhosis). The concentration of sodium and other electrolytes in plasma is reduced by simple dilution; the condition should be suspected if the sodium concentration is below 134 mEq/l, but salt-depletion must be excluded.

### **Salt and water depletion**

This is the commonest electrolyte disorder, occurring with exposure to high environmental temperatures and with fevers, vomiting and diarrhoea, uncontrolled diabetes, and many other conditions. The symptoms include weakness, listlessness, faintness on standing, headache, vomiting, thirst, and in severe cases profound prostration and mental disorientation. Oliguria is always present, except in cases due to uncontrolled diabetes, the administration of diuretics and Addison's disease.

The physical signs include dry mouth, soft eyes and inelastic skin. There is marked loss of body-weight, low blood-pressure and a rapid pulse. The urinary findings are those of reduced volume and raised specific gravity (with the exceptions mentioned above), but the specific gravity of the urine is not raised to the same extent as in pure water depletion. Accurate measurement of urinary output and fluid balance are essential to the assessment and management of salt and water depletion and repeated estimation of urinary specific gravity is helpful. Estimation of chloride content of the urine (see p. 176) may be used as a rough guide to salt excretion.

Measurement of plasma electrolytes shows only a slight reduction of serum sodium concentration despite a severe deficit in the body, as the volume of extra cellular fluid is markedly reduced. The blood urea becomes elevated as renal excretion falls, and is a useful indication of sodium depletion, provided that renal disease can be excluded.

### **Salt and water overload**

This is generally a condition induced by ill-chosen therapy, but may also be found in conditions causing failure of excretion of salt and water, such as renal failure, congestive heart-failure and cirrhosis.

The symptoms are dependant oedema and occasionally breathlessness. These are usually accompanied by heart-failure, with raised jugular venous pressure and crepitations at the lung bases, and there is an increase in body-weight. The urine is unremark-

able, except in conditions associated with failure of excretion, in which the urine specific gravity is usually close to that of the plasma (1012) and the urine volume is reduced.

Management again requires accurate recording of fluid-balance, and daily measurements of body weight are helpful. Estimation of the sodium-concentration in the plasma is of little value, as the extracellular compartment simply expands to contain the increased content of salt. The blood urea is normal unless renal failure is present.

### Potassium depletion (Hypokalaemia)

The gastro-intestinal juices contain a considerably greater concentration of potassium than does the extra-cellular body fluid, and loss of potassium in diarrhoea or vomiting, or through intestinal fistulæ, are the common causes of this disorder, which may be combined with water depletion and salt depletion. Abuse of aperients may also cause prolonged potassium loss, and malabsorption in small bowel diseases is another of the conditions which may lead to hypokalaemia. Excessive loss of potassium in urine occurs with the use of diuretics, and may also arise in certain renal diseases, diseases of the adrenal cortex and following prolonged diabetic ketosis.

The symptoms of potassium depletion are those of muscular weakness, abdominal distension and constipation, later leading to vomiting, depression, and impairment of intellectual activity. Physical signs include abdominal distension, associated with reduced bowel-sounds, depressed tendon-reflexes, the presence of extrasystoles or other cardiac arrhythmias, and in severe hypokalaemia the urine specific gravity is relatively fixed at or near 1012. The urine is usually acid to litmus, and the urine volume is relatively large.

Estimation of electrolytes in plasma is helpful, but it must be remembered that the bulk of the body-potassium is not to be found in this compartment. A concentration of potassium in plasma below 4 mEq/l is abnormal, and the level may fall below 2 mEq/l. The plasma bicarbonate is raised (above 30 mEq/l), for reasons related to acid-base balance (page 335). The electrocardiogram is abnormal in potassium depletion, being of low voltage with flat T-waves and usually a prominent U wave following the T, and this is a useful test, since it reflects the *intracellular* potassium.

Balance-studies, with estimation of intake and urinary loss of

potassium, are often essential for the management of potassium-depletion, and careful assessment is essential in every patient because of the dangers of potassium excess.

### Potassium excess (Hyperkalaemia)

This is a condition which most commonly arises through failure of excretion of potassium by the kidneys, but it may also be caused by injudicious therapy. Alterations in distribution of potassium in the body, leading to increase in extracellular potassium, may occur with extensive trauma or other conditions involving breakdown of tissues.

The symptoms of potassium excess are few. The patient may be anxious or may be lethargic. Sudden death may arise from cardiac arrest. Physical signs are confined to the infrequent occurrence of cardiac arrhythmias, and in patients suffering from diseases likely to cause it, the condition must be diagnosed by special investigation. The electrocardiogram is abnormal, with very tall T waves in most leads, and the serum potassium concentration rises above 5.5 mEq/l, often reaching 8 mEq/l before death occurs. Balance data are not usually required once this high plasma concentration has been found and the electrocardiogram recorded.

## ACID-BASE BALANCE

The regulation of the acidity or pH of the body is a function of the buffer systems of plasma, and, even more important, of the kidneys and lungs. The latter ordinarily play a passive role as a route of excretion of carbon-dioxide, a function governed by the pH and bicarbonate content of the plasma, but respiratory failure or hyperventilation may cause disturbance of blood pH through accumulation of carbonic acid (respiratory acidosis) or over-excretion of carbon-dioxide (respiratory alkalosis).

The kidneys play the major role in regulation of body pH through excretion of hydrogen ions, which is linked with the production of ammonia in the renal tubule and the excretion of phosphate. In renal disease, failure of excretion of acid may lead to increased acidity of the blood (low pH) without compensatory changes in the urine, but in other forms of acidosis the kidneys attempt to correct the disorder by the excretion of an acid urine. Alkalosis represents a more difficult situation for the kidneys to correct. Whilst the urinary pH can be pushed as low as 4.6 in acidosis, the kidneys are

capable of producing only a mildly alkaline urine with pH up to 8.0. Furthermore, if alkalosis should be associated with potassium deficiency (which is usually the case in prolonged vomiting), the capacity of the tubules to excrete an alkaline urine is even further impaired. In potassium deficiency itself, loss of potassium from within cells is accompanied by a shift of hydrogen ions from extracellular to intracellular fluid, leading to acidosis within the cells and a fall in hydrogen-ion concentration in plasma and interstitial fluid—that is, a rise in pH, or extracellular alkalosis.

The changes in plasma and urine in acidosis and alkalosis of respiratory origin and other forms is shown in the table below, but it should be noted that the extracellular alkalosis and intracellular acidosis of potassium depletion does not fit the pattern described for primary alkalosis, in that the urine is acid. The body must excrete a substantial quantity of acid derived from metabolism of food every day, and failure of acid secretion in renal disease rapidly results in severe acidosis in the body, even though the urine excreted usually remains only mildly acid.

	<i>Respiratory acidosis</i>	<i>Respiratory alkalosis</i>	<i>Non-respiratory acidosis</i>	<i>Non-respiratory alkalosis</i>
Plasma pH	Down	Up	Down	Up
Plasma bicarbonate	Up	Down	Down	Up
Urine reaction	Acid	Alkaline	Acid	Alkaline

The clinical picture in *acidosis* (other than respiratory acidosis) is of deep sighing respirations (Kussmaul breathing), and in all forms of acidosis there is a tendency to warm extremities and a rapid pulse. Headaches and some degree of depression are common, and later there may be clouding of consciousness.

The physical signs in *alkalosis* include excitability, twitching, fits, and tetany. In chronic alkalosis, marked personality change may occur, which can be mistaken for psychosis—hypomania or paranoia.

Diagnosis in every case depends on accurate assessment of conditions which may lead to disturbed acid base balance, and careful interpretation of the plasma electrolyte and urine acidity changes. Measurement of blood pH is occasionally required for accurate quantitative assessment of the deficit of acid or alkali, but is not usually necessary for diagnosis.

## DISTURBANCE OF CALCIUM AND MAGNESIUM METABOLISM

### Hypercalcaemia

Hypercalcaemia may arise from a number of causes, including Vitamin D and calcium medication, mobilization of calcium from the bones in hyperparathyroidism, carcinomatosis, and multiple myeloma, in sarcoidosis, and occasionally in renal disease. Clues as to the presence of any of these states must be sought in the history of a patient suspected of suffering from hypercalcaemia.

Symptoms of hypercalcaemia are depression (often mistaken for psychosis), nausea, anorexia and vomiting, abdominal distension and constipation, and frequently nocturia and polyuria due to impairment of renal function. Cardiac arrhythmias may occur, with extrasystoles, paroxysmal tachycardia and fibrillation. The physical signs follow from these symptoms.

Measurement of the total serum calcium is essential to diagnosis, and in most laboratories the normal range falls between 8.6 and 10.5 mg. per cent (approximately 5 mEq/l). Concentrations of above 11.0 mg per cent produce symptoms of hypercalcaemia, and concentrations above 12 mg per cent are dangerous.

### Hypocalcaemia

Hypocalcaemia may arise through deficiency of Vitamin D and calcium, through malabsorption in small-bowel disease, through disorder of intermediary metabolism in hypoparathyroidism, or to rapid deposition of calcium in bones after the removal of a cause of osteoporosis. Inquiry into the possibility of any of these conditions is therefore important in taking the history. Symptoms are generally those of irritability and vague ill-health, but in acute or severe cases paraesthesiae, twitching, tetany and convulsions may occur. Diagnosis depends on the demonstration of a low serum calcium concentration (below 8.6 mg. per cent in the fasting state).

### Magnesium metabolism

Disturbance of magnesium metabolism generally accompanies calcium disorders. However, magnesium depletion may occur alone in patients with severe diarrhoea and with gastro-intestinal fistulae, and it also occurs in severe nutritional deficiency in

Kwashiorkor. The clinical picture is one of depression, marked muscular weakness, vertigo, and a tendency to convulsions, tetany, and stupor in severe deficiency. The diagnosis is confirmed by the finding of a concentration of magnesium in plasma of less than 1.5 mEq/l. Proof of total body deficiency depends on observing the response to the administration of 100-200 mEq of magnesium over some four hours.

## **13 CLINICAL EXAMINATION OF CHILDREN**

The clinical examination of young children is an art which must be learnt by experience. However, children who object very strongly to being examined usually do so because they are afraid, but an adequate examination can often be achieved even then by patience and a gentle approach. The child and his mother should be greeted in a friendly manner and the child offered a selection of toys suitable to his age. The *history* is then taken from the mother according to the scheme set out on p. 11. While talking to the mother, the examiner should observe the patient and take note of certain points. Does the child look well or ill? Is there anything unusual about the facies? Are there any obvious physical deformities? Is the child well nourished or wasted? Is there any difficulty in breathing?

### **The General Examination**

The child should now be at ease and getting used to the strange surroundings. This is the time to record the respiration and pulse rates. The *respirations* can usually be counted by watching the movements of the chest or abdomen, which moves much more with respiration in young children than in adults. The average rate in a normal newborn child is 40 respirations per minute; by the second year it has fallen to 25 or so, and by the fifth year to about 20. A raised respiratory rate in a child at rest usually indicates some disorder of the respiratory or cardio-vascular systems. The normal pulse-respiration ratio is 3 or 4 to 1.

The *pulse* is counted in young infants by palpation or auscultation of the chest, in older children by palpation at the wrist. The average pulse-rate at birth is about 130 per minute, at one year 110, at three years 100, at eight years 90, and at 12 years 80. During sleep the pulse-rate falls about 10–20 beats per minute. The normal limits for pulse-rates are wide. Tachycardia is common, and may be due to crying, excitement, exercise, and fever as well as

to various diseases. Bradycardia is rare, and usually indicates some cardiac abnormality. Sinus arrhythmia is present in almost all children, but other irregularities of rhythm are uncommon even in the presence of cardiac disease.

Examination should now proceed by the usual methods of inspection, palpation, percussion and auscultation; but no set routine can be followed, and the examination is by regions rather than by systems. Each patient will, by his reaction to the various procedures, dictate the order of examination and even the position in which he will be examined. Young infants are usually examined on the mother's lap, older children may not be prepared to lie down on a couch and can be examined standing at the mother's side. Most children can be persuaded to have their clothes removed, although they are often modest and should be allowed to keep their pants or knickers on.

It is important to be gentle and to have warm hands. Begin by looking at and feeling the child all over. Observe the general state of development and nutrition. Is the skin dry or moist, and is there the normal degree of elasticity? Is there a rash present? The shape of the chest and abdomen should be noted. The abdomen is normally rather protuberant in young children. Feel the head and the *anterior fontanelle*: this closes normally between 15 months and two years, and delayed closure may be due to rickets or hydrocephalus, whilst too early closure occurs in some forms of cerebral disease. The posterior fontanelle is always small, and closes by the second month. The degree of tension of the anterior fontanelle is important. In health it pulsates and is in the same plane as the rest of the surrounding skull. A depressed fontanelle is a sign of dehydration, and a tense bulging fontanelle indicates raised intracranial pressure. The fontanelle is normally tense when the child is crying.

The *shape of the head* should be noted. It may be abnormally shaped, owing to premature fusion of the sutures. It is globular in hydrocephalus, and is often asymmetrical in normal infants who tend to lie persistently on one side.

The *limbs* should be examined next. Look for swelling and tenderness, which may be due to osteomyelitis. Examine the wrists for widening of the epiphyses of the radius and ulna, which is a sign of rickets. Look for tenderness or limitation of movement of the joints, and for swelling around or in the joints. Examine the inguinal, epitrochlear, axillary, and cervical lymph nodes for

swelling and tenderness. Feel for the femoral pulses on both sides: absent femoral pulses suggest coarctation of the aorta. Finally inspect the spine for the presence of deformity, and test the range of flexion and extension.

One should next proceed to examine the *abdomen*. This is the one part of the body which it is impossible to palpate satisfactorily in a child who is crying or resisting. Small infants can be given a feed from a bottle to stop them crying, but with an older child one must wait until he settles down before attempting to palpate the abdomen. Children from about one to three years old will often refuse to be examined lying down, but in this age group it is often possible to palpate the abdomen from behind when the child is standing on the mother's lap and looking over her shoulder.

Palpation should be gentle and light. The liver edge can be felt quite easily and in young children normally extends down to 2 cm. below the costal margin. The spleen when enlarged can be felt below the left costal margin. Slight enlargement of the spleen is common in children with infections of all types. Fecal masses can be felt in constipated children, and a full or distended bladder presents as a firm mass arising out of the pelvis. Abdominal tenderness is best detected by watching the child's facial expression during palpation.

The *thorax* should be inspected for deformity of the chest wall and for intercostal or subcostal indrawing, which may indicate some type of respiratory obstruction. Look also for the grunting respirations of the child with pneumonia or other respiratory disease. This is due to a reversal of the normal respiratory rhythm. The grunting expiration is followed by inspiration and then a pause. The thickening of the costo-chondral junctions ('rickety rosary') which occurs in rickets can be seen and felt. Palpate the anterior chest wall for the cardiac impulse and for thrills. In children under the age of six or seven years the cardiac impulse is normally in the fourth intercostal space, just to the left of the mid-clavicular line. In older children it is usually in the fifth space in the mid-clavicular line. Vocal fremitus is not a sign of great value in children.

Percussion of the chest should be light and in small children can be direct, that is to say the chest wall is tapped directly with the percussing finger without the use of a pleximeter finger. The chest is much more resonant in children than in adults.

A stethoscope with a small bell chest-piece is suitable for general

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auscultation of a child's chest. Listen for the breath sounds and for adventitious sounds. Because of the thin chest wall, breath sounds are louder in children than in adults, and their character is more like the bronchial breathing of adults (puerile breathing). Upper respiratory infections frequently give rise to loud coarse rhonchi, which may be conducted down the trachea and main bronchi.

The first and second heart sounds may be split in any area in children, and the pulmonary second sound is normally louder than the aortic second sound. Venous hums and functional systolic murmurs are often heard in normal children.

In older children examination of the *nervous system* can be carried out in the usual manner. The neurological examination of newborn infants is a specialized technique, beyond the scope of those without experience in this field. In children of an intermediate age, the extent of the neurological examination depends on their age and willingness to cooperate. Certain special points should be mentioned. If the child can walk, look for abnormalities of gait and the presence of a limp or ataxia. Note any abnormal movements. Tics or habit spasms are repetitive but purposeful movements, such as turning of the head or shrugging of the shoulders. Choreaiform movements are coarse, involuntary, purposeless jerks which follow no particular pattern. These are best demonstrated by asking the child to hold out his arms in front of him. In this position the child with chorea adopts a characteristic posture, with the wrists in flexion and the fingers in hyperextension. Co-ordination is tested by some modification of the finger-nose test, for example reaching out to touch a toy held in the examiner's hand.

Muscle tone and muscle power should be assessed. The child with marked hypotonia will slip through one's hands when picked up under the armpits. Meningeal irritation or spasm of the spinal muscles is detected more readily by resistance to passive flexion of the head and neck (neck stiffness) than by testing for Kernig's sign.

Tendon reflexes are often difficult to elicit in normal children, and some time may be needed to find the correct position of the limb for this purpose. The plantar responses are extensor in normal infants up to the age of about 18 months, and the persistence of an extensor response after the age of 2 years indicates an upper motor-neurone lesion.

With a little ingenuity, most of the cranial nerves can be tested.

For example, by getting a baby to follow a bright object moving in various directions the eye movements can be studied and the presence of nystagmus noted. Examination of the fundi is particularly difficult in young children, as they cannot fix their gaze. Infinite patience is needed for this manoeuvre, as it entails waiting with the ophthalmoscope in position for fleeting glimpses of the optic disc. Forceable attempts to keep the eyes open only make the procedure more difficult.

Gross degrees of deafness can be detected by observing the child's failure to respond to simple sounds, such as a squeaker or a spoon rattled in a cup, made outside his field of vision. More accurate hearing tests can be carried out by experts even in children under six months of age.

The testing of sensation and the more elaborate functions of the central nervous system is difficult in children, but fortunately not of great importance.

For the child the most unpleasant examinations are those of the *ears, nose, mouth, and throat* and these should be left until last. With skill, gentleness and patience, even these procedures can usually be carried out without making the child cry. Start with the nose, which need only be examined superficially. By placing a shiny surface, such as a mirror, under the nostrils, patency of the nasal airways can be judged by the size of the area of clouding. With a good light, the appearance of the mucous membrane of the anterior nares can be inspected. Proceed next to examine the external auditory meati and tympanic membranes. Allow the child to see and handle the auriscope and speculum, using the instruments as a toy in a simple game for a few moments. If, in spite of these preparations, the child still resists, he will have to be held by his mother. Sit the child on her lap facing to one or other side, and get her to hold the child firmly with one arm around his head and the other around his upper arms and shoulders. Held in this way, the child can be kept still long enough for the ear-drums to be inspected.

The mouth and throat are examined in similar fashion. A co-operative child can be encouraged to 'show his teeth' and thus open his mouth without the use of a spatula, which so many children dread. The uncooperative or very young child must be held by the mother, as for the examination of the ears, but in this case the child sits facing the examiner. The use of a spatula may be unavoidable. Force it gently between the teeth and on to the

tongue, which is then depressed. Note the state of the teeth, the tongue, and the mucous membrane of the mouth. Look for Koplik's spots, the pharyngeal lesions of chicken pox, and the white patches of thrush. Inspect the tonsils and the pharynx. Look for streaks of mucopus on the posterior pharyngeal wall (post-nasal drip).

### Special Examination

Certain additional points in examination are now considered briefly.

**1 Measurements.** Measurements of weight and height are important in the examination of children. Height can be measured only in children over the age of about two years. Below this age supine length can, if necessary, be measured roughly with a tape measure. All measurements should be made under standard conditions, and children should be weighed unclothed. One of the particular features of childhood is that it is a prolonged period of growth, the pattern of which may be adversely affected by many disturbances of health. Heights and weights should be compared with those of healthy children of similar age and build and for this purpose the percentile charts (pp. 381-386) are invaluable. Serial measurements over a period are essential in assessing changes in growth rates. As a rough guide, the average weight of normal children can be taken as 7 lb. (3.2 kg.) at birth, 14 lb. (6.4 kg.) at 6 months, 21 lb. (9.6 kg.) at one year, 28 lb. (12.8 kg.) at two years and 49 lb. (22.4 kg.) at 7 years ('rule of seven'). However, it can be seen from the percentile charts that there is a wide range above and below the average.

In infants under the age of two years, the head circumference should be measured. The standard measurement is the occipito-frontal circumference, which is the largest circumference of the head. The average head circumference at birth is 14 inches, at 3 months 16 inches, at 6 months 17 inches, at one year 18 inches and at 2 years 19½ inches. The diagnosis of hydrocephalus should be suspected when the rate of growth of the head is greater than normal for the age and size of the infant.

**2 Blood pressure.** Abnormalities of blood pressure are uncommon in childhood, and measurement of blood pressure is a distressing examination for some young children. Consequently this procedure is sometimes omitted from the general examination, but it must be

carried out in all cases of suspected cardio-vascular disease. Blood pressure readings are best obtained in most children after the main examination is over and the child is partially dressed. Allow the child to see and play with the cuff, and give some simple explanation of what is going to happen. The size of cuff is most important if accurate readings are to be obtained, and a variety of sizes are available. The inflatable bag should be long enough to encircle the full circumference of the arm and should be of a width roughly equal to half the length of the upper arm. In small children and infants it may not be possible to determine the blood pressure by auscultation, and the pulse can be palpated to obtain the systolic pressure. In babies the flush method may be used. The arm is held up and tightly bandaged to exclude the blood to the level of the cuff which is then inflated. The bandage is then removed to reveal a white limb. The pressure in the cuff is slowly reduced. The point at which the skin flushes is an approximate indication of the systolic pressure. The blood pressure in the legs must be determined in suspected cases of coarctation of the aorta. The average level of blood pressure in the arms is 80/50 in the newborn, 85/60 at 4 years, 95/65 at 8 years, 100/70 at 10 years and 110/75 at 13 years.

**3 Temperature.** The taking of the temperature is not an essential part of the examination of children. Fever is a very common finding in children and may be due to excitement, exercise, and minor infections as well as to severe infections and other serious illnesses. Small infants often respond to infections with low temperatures. The temperature may be taken either before or after the physical examination. Oral temperatures are taken in children over the age of about 6 years. In smaller children, between 2 and 6 years, the thermometer can be placed in the axilla or groin. Rectal temperatures are taken in infants. The temperature in the axilla or groin is about 1° F (0.5° C) lower and in the rectum about 1° F higher than the oral temperature. The temperature of normal children varies between 1° F below and 1° F above the average. Rapid rises of temperature to 103° or 104° F (39.5° or 40° C) are sometimes associated with febrile convulsions in young children.

**4 Rectal examination.** Most children find rectal examination extremely unpleasant, and small infants experience some pain from the procedure even when the little finger is used. One must use one's judgement, therefore, in deciding whether this examination should be inflicted on a child. Anal fissure is a common

condition in childhood, and can be detected by close inspection of the anal canal, the buttocks and anal orifice being gently held open with one hand on either side.

**5 Stools.** The stools of normal breast-fed infants may be loose and green or pasty and yellow. Infants fed on cow's-milk preparations pass stools of a paler yellow colour and of a much firmer consistency. The character of the stool in older children is more variable than in adults.

**6 Urine.** Special techniques are required for the collection of urine specimens, in infants who have not yet acquired sphincter control. In boys the penis may be inserted into a sterile test tube which is then held in place by adhesive strapping. In girls it is necessary to use a specially made plastic container, the mouth of which is adhesive and shaped to fit on to the labia.

**7 Developmental Examination.** An important part of the clinical examination of young children is the assessment of developmental progress, which is largely a function of maturation of the nervous system. In certain cases a detailed developmental examination will have to be undertaken, but this is a specialist procedure. In a general examination only a few basic observations need be made. The main fields of development are motor activity, speech, vision, hearing, and behaviour. Assessment of a child's attainments in these fields will have been made in the preliminary observation of his attitude and behaviour and in the subsequent examination of the various systems, particularly the nervous system. A representative selection of the stages of development are referred to as 'milestones'. The following is a list of milestones with the average age at which they are reached by normal children. There is of course a wide variation in the rate of development in individual children.

### Milestones of Development

Smiles (6 weeks)

Holds head steady (3 months)

Turns head to sound (4 months)

Reaches out and grasps object (5 months)

Maintains sitting position (7 months)

Transfers objects from one hand to the other (7 months)

- Stands with support (9 months)
- Crawls (11 months)
- Says 2-3 single words with meaning (12 months)
- Walks with support (13 months)
- Holds cup and drinks (15 months)
- Builds a tower of 3 cubes (18 months)
- Says 2-3 word sentences (2 years)
- Dresses himself (3 years)

### Examination of the newborn

Detailed examination of the newborn infant requires special techniques and skills. It is possible, however, to obtain important information by a few simple observations, which will now be described.

The newborn infant must be examined with all his clothing removed. Careful inspection will reveal any anatomical defects, such as cleft lip, abnormalities of the limbs, or spina bifida cystica. In looking for anatomical defects, remember to examine the palate (for clefts), the anus (which may be imperforate), and the external genitalia. The testes can usually be felt in the scrotum in baby boys.

Observe the colour of the skin. Peripheral cyanosis is a common finding in newborns, but central cyanosis indicates cardiac or respiratory disease. So-called physiological jaundice is present after the first 48 hours in most premature babies and in some full-term babies. Severe jaundice within the first 48 hours is usually due to some haemolytic disorder. Look for haemangioma in the skin. These may be flat capillary lesions or raised capillary-cavernous lesions (strawberry birthmark). In postmature babies the skin may be dry, cracked and peeling. Patchy papular lesions which fade rapidly are common and of no serious significance (*erythema toxicum*).

Breast enlargement, often with exudation of a milky fluid from the nipples, is frequently seen in newborn infants of both sexes. This is due to maternal hormones and is of no consequence.

The head should be inspected, palpated and measured (occipito-frontal circumference). There is considerable normal variation in the size of the fontanelles and in the width of the suture lines. A cephalhaematoma is a subperiosteal haematoma which appears a few days after birth as a large cystic swelling limited to the area of one of the bones of the skull-vault. The *caput succedaneum* is an

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area of oedema of the scalp over the presenting part of the head. It pits on pressure and is not fluctuant.

It is essential to examine the hips of all newborn infants, in order to detect actual or potential dislocation. The infant is laid on his back with hips and knees flexed. The examiner grasps the thighs and rotates them internally and externally with a twisting action of his wrists. An unstable hip joint is indicated by a pronounced click as the head of the femur comes out of the acetabulum and slips back again.

## **14 SOME LABORATORY INVESTIGATIONS**

In this chapter the indications for certain bacteriological and other laboratory investigations will be considered, together with the methods for collecting materials for examination. This brief account in no way replaces pathological and bacteriological textbooks, which must be consulted for further details.

### **I COLLECTION OF SPECIMENS**

It is essential that all specimens sent to the laboratory should be legibly labelled with the patient's surname and christian name, age and sex. It is also important to indicate the exact nature of the material sent, its source, and the investigations requested. The name and telephone number of the doctor sending the specimen should be included; and, in the case of hospital patients, the patient's hospital record number.

It is generally desirable, and frequently essential, that the pathologist should also be given a note of the duration of the illness, the tentative diagnosis, and the clinical state of the patient. In this way the examination of the specimen in the laboratory is facilitated and the pathologist is in a better position to indicate the possible significance of his findings. The result of pathological investigations must finally be assessed in relation to the clinical condition by the physician in charge of the patient, but it will often be valuable to have the opinion of the pathologist in the interpretation of laboratory findings. One of the most valuable functions of the laboratory is to test the sensitivity of organisms of all kinds to antibiotics. Good antibiotic treatment demands close collaboration between clinician and pathologist, and a note of current antibiotic therapy must always be sent with specimens for bacteriological examination.

It is essential that specimens reach the laboratory fresh and correctly taken into the correct kind of containers. Specimens

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are best taken by hand to the laboratory as soon as they are obtained, but they can, if necessary, be sent by post (letter post only), provided they are suitably packed and labelled 'Fragile, With Care' and 'Pathological specimen'. Such specimens must be placed in a sealed inner container, and then packed in a wooden or metal box containing sufficient absorbent material to soak up all the liquid contents if the inner container is broken.

Suitable containers are best obtained from the laboratory that is going to make the investigations. All containers must be perfectly clean and preferably sterile. This is of course essential for bacteriological specimens. Containers for blood should be completely dry. All containers must have properly-fitting lids or caps. It is usually essential that the correct container should be used for each particular investigation (e.g. particular anticoagulants are necessary for particular chemical or other tests on blood). It is also desirable that the amount of material specified for a particular container should be placed in that container, and in any case no more than the amount specified.

All syringes and needles must be sterile and should either be of the disposable variety or be dry-sterilized in a laboratory.

**Venepuncture.** A piece of rubber tubing is used as a tourniquet and applied round the upper arms over the middle of the biceps, so as to impede the venous but not the arterial flow. The skin at the bend of the elbow is 'painted' with 2 per cent iodine in spirit or cleaned with spirit. The skin is rendered tense by the operator's left hand; the syringe with the needle attached is held in the right hand and almost parallel with the patient's arm; the patient is asked to 'make a fist', and then the needle with the bevel upwards is inserted into a prominent vein—the median basilic is usually convenient, and the needle is pointed in the direction of the blood flow. The required amount of blood is then drawn up into the syringe and the tourniquet is removed before the needle is withdrawn, as otherwise a haematoma may form. For some purposes it is necessary to remove the tourniquet as soon as the needle enters the vein, so that free-flowing blood is withdrawn. As soon as the needle is withdrawn a swab is placed on the puncture site and the patient is instructed to hold his forearm firmly flexed against his arm for a minute or so.

Occasionally a vein in the forearm or wrist may prove more convenient than one at the elbow.

Blood obtained by venepuncture should immediately be placed in a container suitable for the purpose for which it is to be used. The needle should first be removed from the syringe, since forcing the blood through the needle may cause haemolysis. Appropriate containers for particular investigations should be obtained from the laboratory, since, when unclotted blood is required, different anticoagulants are needed for particular purposes.

Heparin and sequestrene (EDTA) are the most generally useful anticoagulants. They can be used for most haematological investigations as well as for most simple chemical tests, with the exception of blood glucose, for which special bottles containing sodium fluoride are necessary.

For blood groups and serological investigations blood should be taken into a dry sterile bottle or tube. If the specimen has to be sent to the laboratory by post, it is best to wait till the blood has clotted. Some serum should then be removed with a sterile needle and syringe, and this serum should be sent separately, together with the blood clot.

## II BLOOD CULTURE

*Blood culture* is indicated when bacteraemia is suspected. In this condition bacteria are present in the circulating blood, reaching it from a poorly-localized focus of infection. Bacteraemia may be suspected in local pyogenic infections when the temperature and pulse-rate fail to settle down. Typhoid or paratyphoid bacilli are always present in the blood in the early stages of enteric fever, and the infecting organisms may be isolated by blood-culture in cases of undulant fever, infective endocarditis and lobar pneumonia. In cases of pyrexia of unknown origin, the isolation of an organism may establish the diagnosis, and samples of blood should, if possible, be taken before any antibiotic is administered. They are best taken when the temperature is rising, and are unlikely to give a positive result when the temperature is normal or sub-normal.

Blood cultures are often contaminated when the cultures are inoculated by the inexpert. This operation is therefore best undertaken by the pathologist; but in any case the strictest aseptic precautions should be taken. A clean venepuncture is essential. The needle and the bottleneck should be flamed before the blood is squirted into the bottle, through the needle in this case, and both

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the cap and the neck of the bottle are flamed before the cap is replaced. The blood is at once placed into suitable tubes or flasks of medium. It may be desirable to ascertain the number of living bacteria in the blood, and for this purpose the specimen should be mixed with an equal volume of a sterile solution of 0·3 per cent sodium citrate in 0·6 per cent sodium chloride. Coagulation is thus prevented, and known quantities of blood can then be incorporated in a suitable medium.

Success in the demonstration of bacteria which are present in the blood-stream depends on the number present, the amount of blood taken for culture, the use of suitable media, and the conditions of cultivation. For most purposes the volume of medium used should be at least ten times the amount of blood added, e.g. 10 ml. of blood to 100 ml. of culture medium. The medium may with advantage contain trypsin, or some other substance, to prevent the clotting of the added blood and to destroy the anti-bacterial properties. Where bacteria are few in number, several days may be required before they are detected in culture.

It is usually advisable to put up anaerobic cultures as well as aerobic ones. Primary anaerobic culture can be carried out by using a semi-solid medium containing thioglycollic acid. For slope or plate cultures the simplest device is the jar of McIntosh and Fildes in which the oxygen is removed by reacting with a stream of hydrogen with a catalyst.

In suspected cases of Weil's disease, the specimen of blood should be added to citrate to prevent clotting. The casual organism (*Leptospira icterohaemorrhagiae*) will not grow in ordinary culture media, but may be detected by special cultural methods or by animal inoculation.

Bacteraemia is usually present in infections with *Salmonella typhi*, *Salmonella paratyphi A* and *B*, *Brucella abortus*, *Brucella melitensis*, and the leptospira of Weil's disease, and may be a complication of infections due to streptococci, staphylococci, pneumococci and meningococci. Other organisms such as *Escherichia coli* and *Cl. welchii* are occasionally found in the blood-stream.

The identification of an organism isolated by blood culture may serve to determine the nature of an infective condition and also as a guide for specific treatment. Once an organism is isolated by blood culture or other methods, and specific treatment is contemplated, the sensitivity of the organism to antibiotics,

both qualitative and quantitative, should if possible be determined before treatment is begun; though in some instances it may be justifiable to begin treatment without bacteriological control.

### III CEREBRO-SPINAL FLUID

Examination of the cerebro-spinal fluid by bacteriological methods should be made in any patient in whom meningitis is suspected.

The technique of lumbar puncture has already been described. In cases of meningitis the number of cells is always increased and, except in meningitis of tuberculous or syphilitic origin and in cases of meningitis due to virus infection, the fluid is usually turbid and may be frankly purulent. Films should be made from the fluid, after centrifugation if necessary, and stained by Gram's and by Ziehl-Neelsen's methods. Bacteria may be recognized in films, and the results of this examination will indicate what cultural methods are to be adopted for the isolation and identification of the infecting organism.

In cases of tuberculous meningitis, the bacilli are usually scanty and may be found in films of the centrifuge deposit only after prolonged search. Better results may be obtained if the fluid is allowed to stand at room-temperature for some hours, when a delicate cobweb-clot of characteristic appearance frequently forms. A film made from the clot and stained by Ziehl-Neelsen's method will usually show acid-fast bacilli, if a careful search is made. The organism requires several weeks before visible growth appears on suitable media, and as the test by animal inoculation also takes three to six weeks, these two methods are seldom of practical clinical value in cases of tuberculous meningitis. All fluids containing an excess of lymphocytes should be examined for their glucose content. A lymphocytic fluid with a glucose-value below 50 mg. per 100 ml. should be taken as an indication for commencing treatment for tuberculous meningitis.

Meningitis may also be due to the meningococcus, pneumococcus, streptococcus, staphylococcus, *Haemophilus influenzae*, and, less commonly, to other organisms. In these instances, cultural methods should always be used to confirm and supplement the results obtained by microscopic examination of stained films of the fluid.

#### IV SPUTUM

Bacteriological examination of the sputum is indicated in any inflammatory condition involving the trachea, bronchi, or lungs.

The specimen is best collected first thing in the morning. The mouth should be washed out with warm water, so as to avoid, as far as possible, oral contamination and excessive mixture with saliva. The specimen should be collected in sterile wide-mouthed metal or glass containers.

The bacteriological examination of sputum may be considered under two headings: (i) for *Mycobacterium tuberculosis*; (ii) for other bacteria.

If the tubercle bacillus alone is being sought, a specimen may be satisfactory even if it is two or three days old. When, however, it is desired to obtain a true picture of the bacterial flora, the specimen should reach the laboratory with as little delay as possible.

**1 Examination for tubercle bacillus.** A mucopurulent portion may be selected and spread fairly thickly on a microscope slide. Only new slides should be used. More satisfactory results are obtained if the sample of sputum is mixed with 5 volumes of 1 in 20 carbolic acid in a stoppered container, which is then thoroughly shaken. The specimen is then allowed to settle overnight and the supernatant fluid is discarded. The remainder is spun in the centrifuge for 5–10 minutes and films made from the deposit. The film should be allowed to dry, fixed by heat, and stained by Ziehl-Neelsen's method. The tubercle bacillus, being acid-fast, retains the fuchsin and shows up red against the blue background.

In young children and infants who habitually swallow their sputum, stomach-washings may be examined.

A single negative examination for tubercle bacilli should not carry great weight, and the examination should be repeated several times in any case of suspected respiratory tuberculosis.

If present in very small numbers, tubercle bacilli may be demonstrated by animal inoculation. This examination, however, entails a delay of 3–6 weeks before a final report can be made. It is also possible to grow tubercle bacilli on certain special media, but a positive report will not be available for from 2 to 4 weeks, as growth is slow. Cultures are kept going for 6 weeks before being considered negative. The results of culture compare very

favourably with animal inoculation; and pathologists are agreed that both methods are immeasurably more sensitive than direct smear examination.

The cultural characteristics enable the tubercle bacillus to be classified as of human or bovine type. This can also be done on the result of certain animal inoculation tests. The great majority of strains isolated are of human origin.

**2 Examination for other bacteria.** For identification of other bacteria, films of the sputum should be stained by Gram's method and cultures prepared on suitable media.

In the bacteriological examination of a child suspected of suffering from whooping-cough, the cough-plate method gives the best results. A Petri dish containing a special potato-extract blood agar is held in front of the patient's mouth during a paroxysm and is placed in an incubator at 37° C immediately afterwards. The examination of the plate within the next few days may reveal the presence of *Haemophilus pertussis*. A better method of isolating this organism is to take a post-nasal swab and plate it on Bordet-Gengou medium that has been covered with penicillin solution.

Sputum from normal individuals may contain various bacteria such as streptococci, pneumococci, diphtheroid bacilli, *Micrococcus catarrhalis*, and *H. influenzae*. Except in cases of respiratory tuberculosis, acid-fast bacilli are very rarely found in the sputum, so that the findings of such organisms in a suspected case of pulmonary tuberculosis is of great diagnostic importance. In attempting to assess the significance of other bacteria found in the sputum of patients suffering from respiratory-tract infections, one may be guided by the presence of pus cells and the relative numbers of various bacteria present.

The examination of sputum for malignant cells is of value in the diagnosis of pulmonary neoplasm. The specimen of sputum must be fresh; it can be stained simply with methylene blue or by other more complicated methods. The neoplastic cells are usually present in clumps of densely-staining round cells or as atypical pear-shaped squamous cells. (Plate XIX)

## V THE THROAT AND NASO-PHARYNX

Bacteriological examination of the throat and naso-pharynx may be desirable (*a*) in inflammatory conditions for identification of the infecting organisms, and (*b*) in healthy individuals, to

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determine the presence of pathogenic bacteria, for example in searching for carriers of diphtheria bacilli, haemolytic streptococci, or meningococci.

Specimens should be obtained by rubbing the surface with a swab of cottonwool wrapped around the end of a strong wire. The swab is kept in a narrow test-tube of stout glass and the other end of the wire may be fixed in the cork stopper. The whole apparatus is sterilized by dry heat and kept ready for use. In taking specimens it is important that no antiseptic should have been used (e.g. as a gargle) for some hours before. Where inflammatory exudate is present, the swab should be rubbed firmly over the affected area, and passed under the edge of the membrane in suspected cases of diphtheria.

The swab used for the naso-pharynx is longer than that used for the throat, and the wire is bent near the extremity. A tongue depressor is used: the wire is passed into the mouth and up behind the soft palate, and then brought into contact with the posterior pharyngeal wall. Care must be taken not to touch any part of the mucous membrane of the mouth.

The method of examination of the material obtained varies according to the nature of the infection. In suspected cases of diphtheria, the swab is used to inoculate a slope of Loesfler's serum and, if desired, other special media containing tellurite. The inoculated media are kept at 37° C and examined for the presence of diphtheria bacilli in suitably-stained films after 6 to 18 hours. The bacteriological findings must be evaluated in conjunction with the clinical condition, and the final verdict must rest with the clinician. In a small percentage of cases of diphtheria the bacteriological report may be negative; *when the clinical picture is that of a diphtheritic infection, it is wiser not to wait for the bacteriological report before administering antitoxin.*

In patients presenting the features of acute tonsillitis a Gram-stained film of the material on the swab may indicate the nature of the infecting organism, and confirmation should be sought by cultural methods using blood-agar and Loesfler's serum.

Vincent's angina may produce superficial ulceration of the throat and gums, and may simulate diphtheria when the lesion is confined to the region of the tonsils. Identification of the causal organisms should be made by the examination of stained films of the exudate. The organisms associated with the condition, a large Gram-negative bacillus and a spirochaete, do not grow on ordinary

culture media. For their demonstration, Gram's stain may be used, but polychrome methylene blue or Leishman's stain give better results.

The identification of meningococci in swabs from the naso-pharynx involves culture on suitable media such as serum or blood agar, and the isolation and identification of the Gram-negative cocci by suitable tests. These organisms tend to die out rapidly in material kept at room-temperature, so that it is essential that naso-pharyngeal swabs should be received and examined in the laboratory as soon as possible after they have been taken.

For the detection of carriers the same general methods are used. It should be noted, however, that for the identification of diphtheria bacilli in such instances, microscopical examination of films from cultures must be confirmed by virulence-tests in the guinea-pig before organisms which resemble diphtheria bacilli can be reported as such. The carrying out of a virulence-test involves a delay of several days before a final report can be received.

A variety of bacteria may be present in the normal throat and naso-pharynx, including diphtheroid bacilli, pneumococci, staphylococci, streptococci, influenza bacilli, and Gram-negative cocci. Apart from the infections due to diphtheria bacilli and the organisms of Vincent's angina, acute inflammation of the throat and tonsils is most commonly due to haemolytic streptococci, although *Staphylococcus aureus*, pneumococcus, and occasionally actinomycetes and *Monilia albicans* may be responsible.

## VI NOSE AND NASAL SINUSES

Examination of material from the nose may be carried out, as in the case of the throat, to identify the organisms responsible for inflammatory conditions or to detect carriers of pathogenic bacteria, e.g. staphylococci, among apparently healthy individuals.

Material from the nose may be obtained by means of a throat swab. In cases of sinus infection, where there is free discharge, a specimen may be blown into a wide-mouthed bottle, or, if the discharge is not free, the sinus may be washed out with sterile saline and the washings sent for examination. The identification of diphtheria bacilli in material obtained from the nose cannot be made by microscopic examination of films from cultures alone and must be confirmed where necessary by a virulence test. For the identification of other bacteria in a nasal discharge, films of

the material should be stained by Gram's method, and the results of the examination of such films should be supplemented by culture on appropriate media.

In swabs taken from the healthy nose, diphtheroid bacilli, staphylococci, pneumococci, and Gram-negative cocci may be present. Inflammation of the nose and accessory sinuses is most frequently due to the pneumococcus, haemolytic streptococcus, and *Staphylococcus aureus*.

## VII FAECES

In any intestinal infection, bacteriological investigation of the faeces may reveal the causal organism. In suspected cases of enteric fever and dysentery this examination should always be made, and is the best method for the detection of typhoid or dysentery carriers.

A loose motion should be obtained, and in the case of suspected carriers it may be necessary to administer a purgative. Of a loose motion 1 ml. is sufficient, and any abnormal portion, e.g. containing mucus or pus, should be selected. The usual sterilized throat swab soaked in faeces and replaced in its test-tube is satisfactory when there is no delay in making the examination. In other cases the specimen should be placed in a wide-mouthed vessel by means of a small metal spoon fitted into the stopper. In suspected cases of bacillary dysentery a satisfactory specimen may be obtained by means of a rectal swab; from material taken from the mucous membrane of the rectum in this way, dysentery bacilli may be recovered more readily than from a sample of stool.

Where the making of cultures for organisms of the enteric and dysentery groups must be delayed for more than a few hours after the specimen has been taken, it is advisable to add to one volume of the faeces two volumes of 30 per cent neutral glycerol in 0·6 per cent sodium chloride solution and to make a thorough mixture. The presence of the glycerol prevents the suppression of the specific organisms by *Escherichia coli* which would otherwise occur.

In the laboratory, films of the specimen are stained by Ziehl-Neelsen's method if the presence of tubercle bacilli is suspected. When the examination of such films shows the presence of acid-fast bacilli this result must be interpreted with caution, for acid-fast organisms other than the tubercle bacilli are sometimes found in the faeces. The identification of tubercle bacilli in faeces will

finally depend on the inoculation of animals or suitable media with a portion of the specimen, treated by one of the methods devised to destroy other bacteria without killing tubercle bacilli.

For the detection of bacilli of the enteric and dysentery groups, a suspension of the faeces should be sown in a selective liquid medium or on a solid medium such as MacConkey's bile-salt lactose agar. The identification of these bacteria depends on their isolation in pure culture and the results of fermentation and agglutination tests. Isolation of the infecting organism in cases of enteric fever or dysentery is not always successful. The results will vary according to the relative number of these bacteria in the faeces, the interval elapsing after the specimen has been obtained and before cultures have been set up, the cultural methods used, and other factors. In dysentery, for example, the specific bacteria may be difficult to isolate after the first few days of an attack, especially if delay between the collection and cultural examination of the specimen occurs. In such cases further specimens should be examined, and after the first week of the disease indirect evidence of the nature of the infection may be obtained by examination of the serum for specific antibodies (*see p. 364*). In the detection of carriers it may be noted that typhoid or dysentery bacilli may appear in the faeces only at irregular intervals and in small numbers. It may therefore be necessary to have many specimens examined, at weekly intervals, before a carrier is detected.

The faeces of normal individuals contain enormous numbers of various types of bacteria, including lactose-fermenting and late lactose-fermenting coliform bacilli, non-haemolytic streptococci, Gram-positive bacilli of the acidophilus type, and various aerobic and anaerobic spore-bearing bacilli. The finding of the bacteria of these groups in pathological conditions affecting the alimentary tract has no aetiological significance.

In cases of suspected amoebic dysentery, a freshly passed specimen of faeces must be examined. A small fragment of blood-stained mucus is placed on a slide, and search is made for rapidly motile amoebae which ingest red cells.

## VIII URINE

In any suspected case of bacterial infection of the urinary tract, examination of the urine should be made. In cases of obstruction due to any cause such as calculus, enlargement of the

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prostate, or urethral stricture, infection is liable to occur, although the symptoms of such infection may be masked by those of obstruction.

Urine affords a suitable medium for the growth of many bacteria, hence it is essential to prevent contamination of the specimen by extraneous organisms, and it is advisable that bacteriological examination should be carried out as soon as possible after the urine has been collected. Because of the danger of introducing infection into the urinary tract, catheters should be avoided unless quite essential. The glans penis or vulva should be thoroughly cleansed with 1 per cent aqueous cetrimide. The urine is then passed into two sterile vessels, the first one of which is rejected in case it has been contaminated. The pathologist should be notified whether or not the specimen is a sterile one and also of the date and hour of collection. (For an improved method of collecting sterile specimens from women, see p. 172.)

If organisms are scanty, the specimen should be centrifuged and films and cultures made from the sediment. Films should be stained by Gram's method, but if there is pus present and the culture is sterile, they should then be stained by Ziehl-Neelsen's method for tubercle bacilli. In staining by Ziehl-Neelsen's method, the film should be treated for one minute with alcohol after decolorizing with acid. By this means the acid-fast smegma bacillus, which is frequently present on the external genitals and may contaminate the specimen, is decolorized. Tubercle bacilli in the urine occur either in small clumps or singly. They are morphologically very characteristic, but are often scanty. In such cases better results will be obtained if the sediment of a 24-hour sample is concentrated in the centrifuge and examined. In cases of suspected renal tuberculosis where pus cells are present in the urine, but no tubercle bacilli can be demonstrated microscopically, cultural methods or the inoculation of the urinary sediment into a guinea-pig may provide evidence of tuberculous infection.

In searching for other bacteria, it is essential that the results of the examination of the Gram-stained films should be amplified by culture, the medium to be used being chosen in accordance with the findings in films. The bacteria can then be identified by their cultural character, fermentation reactions, and, if necessary, serological tests.

In some instances it may be desirable to ascertain the number of bacteria present in a sample of urine. This can be conveniently

done by plating known quantities of the fresh specimen (diluted if necessary) on a solid medium.

Bacterial infection of the kidneys may be due to the tubercle bacillus, *Staphylococcus aureus*, or members of the colon-typhoid-paratyphoid group of bacteria. Pyelitis is most frequently due to coliform bacilli, but cystitis may be due to any of these bacteria, or streptococci, or possibly the gonococcus. In cases of gonococcal urethritis, gonococci are generally to be found in the urinary sediment. In undulant fever due to *Brucella abortus*, or in Weil's disease, the causative organisms may be found in the urine, but as in the case of typhoid and paratyphoid infections, their excretion may be confined to a certain stage of the disease, or may be intermittent.

## IX PUS AND PURULENT EXUDATES

In the examination of pus and purulent exudates one should be guided in some degree by the clinical course of the infection, the tissue or organ affected and, as in the case of actinomycosis (see p. 362), the naked-eye appearance of the fluid which may suggest a particular causal organism.

Pus may be collected by means of a sterile syringe and should be sent to the laboratory in a suitably plugged tube. Only in exceptional circumstances should a swab be used to collect the material.

In general, the examination of pus should be by films and culture. Film preparations should be stained by Ziehl-Neelsen's method if there is any possibility of tuberculous infection, and by Gram's method for other organisms. The preliminary examination of films will be a guide in the planting of cultures. Cultures should in general be made on agar and blood agar. If there is reason to suspect an anaerobic infection, such as tetanus or gas gangrene, a duplicate set of cultures on appropriate media will be set up under anaerobic conditions. Sensitivity to antibiotics of organisms found in pus is now determined as a routine.

The bacteria most commonly found in pus are staphylococci, streptococci, pneumococci and tubercle bacilli. The gonococcus, meningococcus, coliform bacilli, and actinomyces also cause suppurative lesions, while pus formation may occasionally be associated with localized infections by bacteria which do not

usually produce suppuration, such as typhoid and paratyphoid bacilli, *Brucella abortus*, and *Haemophilus influenzae*. In suppurative lesions following wounds, and in those opening onto a mucous surface, more than one type of organism is frequently present.

In gonorrhoea, the ease with which the organism may be detected varies with the stage of the disease and the effect of treatment. In the urethral discharge of acute gonorrhoea in the male, the appearance of Gram-negative intra-cellular diplococci is sufficiently characteristic for diagnostic purposes. In chronic cases where microscopical examination of the discharge or urinary sediment is negative, the organism may be grown on a suitable medium such as agar-containing blood or serum. Gonococci die out rapidly at room-temperature, and inoculated culture media should therefore be incubated at 37° C as soon as possible. If delay is inevitable use should be made of Stuart's liquid medium to ensure preservation of any gonococci present. In the female adult patient, material for examination should be obtained from the urethra or cervix—not from the vagina. In cases of vulvo-vaginitis in children the vaginal discharge should be examined. In chronic cases of gonorrhoea where microscopic and cultural methods have given negative results, the complement-fixation test may yield evidence of infection (see p. 366).

Actinomycosis may be suspected if small yellow granules are present in the pus. These granules can be readily seen if a few drops of pus are added to a tube of sterile saline or distilled water, when they rapidly sink to the bottom. If one of the granules is pressed between a slide and cover-slip, it will be seen to be composed of a central mass of filaments, and homogeneous club-shaped bodies may be seen at the periphery. Films stained by Gram's method show Gram-positive filaments of varying length, some of which show branching. The organism can be cultivated only under anaerobic conditions.

In suspected cases of 'malignant pustule' the anthrax bacillus may be demonstrated in films and cultures made with the exudate from the lesion or the fluid obtained from the blisters which are usually present.

Serous exudates which have any considerable cellular content should be examined in the same way as pus. If the cellular content is scanty centrifugation may be necessary as a preliminary.

## X TESTS FOR SYPHILIS

**Dark-ground microscopy.** In untreated syphilis in the primary stage, careful examination of fluid from the chancre will usually reveal the causal spirochaete, but no treatment, local or general, should be given before the examination. The sore should be cleaned with a sterile swab and saline, and then is squeezed gently until a little serous fluid exudes. If the fluid is blood-stained this should be wiped away until a specimen free from blood can be obtained. The fluid should be taken up in a piece of capillary glass tubing, the ends of which are sealed with a flame and the specimen dispatched to the laboratory. The fluid should be examined as a wet preparation under dark-ground illumination. The spirochaete is recognized by its size and close spirals. Where the equipment necessary for dark-ground illumination is not available the spirochaete may be seen in films stained by Giemsa or Fontana's method, but identification of the spirochaete in such films is not always easy. In the less common extragenital chancres, as for example on the lips or tongue, the examination may be complicated by the fact that other spirochaetes resembling the *Spirochaeta pallida* may be present. As serum tests do not usually become positive until one to three weeks after the appearance of the primary sore, the detection of the spirochaete is the most certain laboratory aid to diagnosis during this period.

**Serum tests.** The Wassermann reaction (p. 365) and the flocculation tests --the Kahn test and Price's precipitation reaction (PRR) -- are used to confirm cases of clinically suspected syphilis and as a routine test in ante-natal women and blood donors. The WR and PRR (the latter done quantitatively) are also used to follow the effects of anti-syphilitic treatment. They are not specific, however, and false positive tests may occur after inoculations and in certain 'auto-immune' diseases. If the tests are positive in the absence of clinical signs of syphilis, they should be repeated. If still positive a verification test should be performed, such as the treponemal immobilization test (TPI). In this the patient's serum is incubated with a suspension of virulent live treponemes and guinea-pig serum as a source of complement. The treponemes are immobilized and killed if a specific antibody is present. This test is of no value in early syphilis. It differentiates however between the 'true' and 'false' positives mentioned above, and

can be used to detect late syphilis when the Wassermann, Kahn and PPR may be negative or equivocal. All these tests, including the TPI, may be positive in other treponemal diseases, such as yaws.

## XI SERUM REACTIONS IN DIAGNOSIS

When a patient becomes infected with bacteria his tissues usually respond by the formation of antibodies. Antibodies are usually specific for the type of infecting organism and so the detection of antibodies against a specific organism may afford evidence as to the exact nature of the infection. The evidence obtained by the examination of a patient's serum for antibodies can in most cases only be presumptive and is never of the same value as the detection of the infecting organism. It is however of greater significance when a rising titre is demonstrated during the course of the illness. Further, as the production of specific antibodies is a response to the infection they will not usually appear during the first week or so of the disease, so that negative serum tests at this time are no evidence against the presence of a suspected infection. In general, the infecting organism is most readily detected before the appearance of specific antibody. The presence of antibody in a patient's serum may be revealed by the agglutination reaction or by the complement-fixation test. Blood is obtained by venepuncture as for culture, except that the blood (5 ml.) is placed in a sterile tube and allowed to clot. The serum is later separated from the clot and freed from blood-cells by centrifugation if necessary.

**Agglutination reactions.** For these tests, increasing dilutions of the patient's serum are mixed in small tubes with suspensions of the type of organisms with which the patient is believed to be infected. The tubes are then placed in a water bath at 46° C for a few hours before being examined. The presence of specific agglutination will be indicated by the appearance of clumps in the bacterial suspension. The agglutinating titre of the serum is the highest dilution which produces visible clumping of the bacteria.

Agglutination tests with the patient's serum may be of value in the diagnosis of infections due to the typhoid and paratyphoid bacilli, *Brucella abortus*, and the spirochaete of Weil's disease.

In assessing the results of agglutination tests in suspected cases

of enteric fever, a previous history of prophylactic inoculation with typhoid vaccine or of enteric infection is important. The agglutination titre of normal human serum for organisms of the typhoid-paratyphoid groups is low, but a higher titre than normal may be present in individuals who have previously been inoculated with typhoid vaccine, or have suffered from typhoid fever. The duration of illness is also important; agglutinins in the serum are strong towards the end of the third week of enteric infection so that, if a result of doubtful significance is obtained by the agglutination test in the second week of a patient's illness, a second examination made a week later will be helpful.

In suspected cases of undulant fever the agglutination test is of great value, as most cases of infection with *Brucella abortus* show a high titre of agglutinins after the first week of the disease.

In testing for agglutinins against the spirochaete of Weil's disease, dilutions of the serum are mixed with a living culture of the organism and the mixtures are examined microscopically by dark-ground illumination. A positive reaction is indicated by the cessation of motility and clumping of the organisms. Practically all cases of Weil's disease give a well-marked positive reaction after the first week of illness.

**Complement-fixation tests.** These tests are based on the fact that serum containing antibody when mixed with a suitable antigen has the power of fixing a measurable amount of complement. Serum to be tested is heated for 30 minutes at 56° C to destroy its own complement. In doing the test, a known dose of complement is allowed to act for a given time with a known amount of antigen and the serum to be tested. At the end of this time the presence or absence of free complement is tested for by adding a known amount of red cells which have been sensitized with a haemolytic antibody. If free complement is still present the red cells are laked and the reaction is negative. If the complement has been fixed no haemolysis takes place and the reaction is positive.

The complement-fixation test in the diagnosis of syphilis is usually known as the Wassermann reaction. A positive reaction may be expected from five to seven weeks from the date of infection, or from one to three weeks after the appearance of a primary sore. The reaction is positive in all cases of untreated secondary syphilis and practically all cases of untreated tertiary lesions. A positive

reaction will be obtained with the cerebro-spinal fluid in all cases of general paralysis, in most cases of cerebral syphilis and, less constantly, in tabes dorsalis. It will be seen, therefore, that, although a negative reaction does not exclude a diagnosis of syphilis, the reliability of the test is such that, in the presence of a negative test, strong evidence will be necessary to uphold such a diagnosis. Remember that a positive reaction may be present in latent cases and, therefore, may be met with in patients suffering from some other disease.

In the complement-fixation test as applied in the diagnosis of gonococcal infection a suspension or extract of gonococci is used as antigen. A positive reaction is not to be expected in the acute stage of gonorrhoea and may never be obtained in simple cases of urethritis. The reaction is generally positive in cases with complications and is of especial value in chronic pelvic disease and in cases of arthritis and tenosynovitis.

In cases of whooping-cough the serum obtained in the third week of the disease and later usually fixes complement in the presence of an antigen made from a suspension of *Haemophilus pertussis*; so that the test is most useful at that stage of the disease when cultural methods are likely to fail.

In cases of hydatid disease the serum usually gives a positive complement-fixation test in the presence of a hydatid antigen. The reaction may, therefore, be of value in suspected cases.

Chronic inflammatory changes in certain tissues, such as occur in Hashimoto's disease of the thyroid, are associated with the production by the patient of antibody against his own cells. Such antibodies are termed '*auto-antibodies*'. This production of antibody can be detected in the patient's serum by a complement-fixation test using the normal tissue as antigen. Immunologists have developed other techniques for the detection of auto-antibodies but in all cases serum from the patient is required, collected as for a Wassermann test. In some cases it is helpful to have a small portion of the affected organ for further examination.

**The Paul-Bunnell test.** The patient's serum is inactivated at 56° C for half an hour. Dilutions are made with normal saline from 1 in 2 to 1 in 128. An equal volume of 2 per cent washed sheep-cells is added, giving final dilutions of 1 in 4 to 1 in 256. The tubes are left overnight at room temperature and next morning each tube is gently inverted to look for agglutination. A positive

result is one in which agglutination occurs in dilutions from 1 in 32 onwards, and may be found in glandular fever (infectious mononucleosis) and in serum sickness. Positive reactions due to the latter condition may be distinguished by the fact that this antibody can be adsorbed out of the serum by minced guinea-pig kidney.

## XII VIRUS AND RICKETTSIAL DISEASES

There are as yet many limitations in the laboratory diagnosis of these conditions; and many of the techniques necessary are complicated, expensive and only available in special centres. In general, investigation by these methods of an outbreak of disease likely to be due to an infectious agent may be fruitful. The investigation of a single patient, thought possibly to be suffering from an unknown virus disease, is less often profitable, unless the investigations are undertaken to confirm a strong clinical suspicion.

Two types of test are available: serological (usually complement-fixation or neutralization tests) and virus isolation.

*Serological tests* are at present much the simpler and more practicable of the two. They require one specimen of 5 to 10 ml. of blood taken (with a clean dry sterile syringe into a clean dry sterile bottle) not more than one week from the onset of the illness, and another specimen taken 2-4 weeks from the onset. If a rising titre of a particular antibody can be demonstrated, such tests are usually diagnostic. It follows that febrile patients in whom virological investigations may be needed later should all have some serum taken on admission to hospital or as soon after the onset of their illness as possible. Such tests are available for the diagnosis of Q fever, psittacosis, lymphogranuloma venereum, influenza A, B, and C, adenoviruses (APC), mumps, lymphocytic chorio-meningitis, and primary herpes simplex.

*Virus isolation* in fertile hen's eggs, in tissue culture, or in animals is a more complicated and expensive affair. Further, specimens which have to be transported any distance must be kept at a low temperature, packed in ice or preferably dry-ice ( $\text{CO}_2$  snow). Clinicians should always consult a virologist or a pathologist specially interested in this subject if they think investigations of this kind are called for in their patients.

### XIII SKIN TESTS

Various skin tests are used as a measure of susceptibility of an individual's tissues to the test substances. This susceptibility is indicated by an inflammatory reaction at the point of application or injection of the test substance; it may indicate a normal susceptibility to toxic material, e.g. bacterial toxin as in the Schick or Dick tests, or it may indicate a state of delayed hypersensitivity or allergy to a bacterial or other protein as in the tuberculin test. In the first instance a positive reaction may be expected in persons who have had no previous contact with the toxic substance, whereas the second (allergic) type of response may be the result of sensitization through previous experience of the test substance.

**The Schick test.** This is a test of susceptibility or immunity to diphtheria toxin. The toxin is so diluted that the test dose is contained in 0·2 ml., and similarly diluted heated toxin is used as control. Glass syringes, graduated in tenths of a millilitre, should be sterilized by boiling and allowed to dry before use; a No. 18 or 20 needle with short bevel should be used. The skin of both forearms should be washed with soap and swabbed with alcohol or ether; when dry the skin is stretched by holding the forearm firmly with the left hand, the injection being made with the right. Introduce the needle almost parallel with the skin and bevel upwards; when the bevel has disappeared inject 0·2 ml. The control heated toxin is injected into one forearm and the unheated toxin into the other. In individuals who are not immune to the toxin a positive reaction will usually appear within 24 hours and will reach its maximum on the 4th or 5th day. Any non-specific reaction due to the protein in the medium from which the toxin was prepared will show within 24 hours on the control arm, but will usually have disappeared by the 4th day when the final reading should be made. By this test it is possible to divide people into two groups, immune and susceptible. By inoculating the susceptibles with toxoid-antitoxin mixtures or floccules it is possible to render them immune. Where this practice has been widely adopted in young children it has been found possible to reduce the incidence of diphtheria to negligible proportions.

**The tuberculin test.** This test is based on the fact that the tissues of a person infected with the tubercle bacillus become sensitized

to tuberculin. The older cutaneous test of Von Pirquet has been largely replaced by the more delicate intracutaneous method of Mantoux and the Heaf test.

**The Mantoux test.** In applying this test 0·1 ml. of a 1 in 10,000 dilution of 'old tuberculin' is injected intradermally into the skin of one forearm, a similar dilution of glycerin broth being injected from another syringe into the other arm as control. A positive reaction is characterized by the development within 48 to 72 hours of an area of erythema and infiltration, 10 mm. or more in diameter, at the site of the injection of the tuberculin. If the reaction is negative after 48 hours a dilution of 1 in 1000 may then be injected and, if this is negative, a further test with 1 in 100 dilution may be made.

PPD stands for purified protein derivative, and this is obtained from a filtrate of cultures of tubercle bacilli grown on synthetic media. PPD is used in the same way as old tuberculin but at two standard strengths based on the weight of the substance present in each dose.

**The Heaf test.** Six puncture wounds are made in the forearm with a special 'gun' through drops of 1 in 3000 old tuberculin (OT). The results are graded from negative to strong positive according to the development and the extent of the erythema and induration.

The value of the tuberculin test is limited owing to the fact that as a person gets older the less likely is he or she to have escaped previous clinical or sub-clinical tuberculous infection. At the age of 1 year, approximately 5 per cent; at 5 years, 20 per cent; at 10 years, 40 per cent; at 15 years, 60 per cent; and at 20 years or over about 90 per cent of individuals in urban Great Britain give a positive reaction to tuberculin. In rural communities the percentage of positive reactions is rather less. In young children a positive reaction will have greater significance, while at all ages a negative reaction will be of value as evidence against a diagnosis of tuberculosis. It must be remembered, however, that in the earliest stages of the disease the reaction may be negative, and in very acute cases or in the last stages, when no response from the tissues can be expected, a negative reaction may be obtained.

**The Kweim test.** Sarcoidosis is usually diagnosed by biopsy of affected tissue. In addition, the tuberculin test is usually negative

and there is hyperglobulinaemia with a reversal of the albumin-globulin ratio. When no biopsy is possible, the Kweim test may be of value. An injection of an extract of sarcoid tissue is made into the skin of the forearm. If the test is positive, a small granuloma appears at the site of injection, and shows typical sarcoid tissue if excised and sectioned four to five weeks later.

**The Frei test.** This test is widely used in the diagnosis of lympho-granuloma inguinale. The antigen consists of pus from a bubo, or brain suspension from an experimentally infected mouse, heated to 60° C for 2 hours. Intracutaneous injection of 0·1 ml. gives rise in an infected subject to an infiltrated inflammatory area, at least 5 mm. in diameter, with a central zone of necrosis. The reaction reaches its height in 48 or 72 hours.

**The Casoni test.** This test is useful in suspected hydatid disease. The antigen is prepared from the fluid or walls of cysts and on injection intradermally gives rise in positive cases to a reaction of the wheal and erythema type. The reaction appears in 5 or 10 minutes and reaches its maximum within about an hour. In some patients a delayed hypersensitivity type of reaction can also occur.

**Other skin tests.** In the investigation of patients suffering from allergic states, such as asthma or hay fever, the sensitiveness to various animal or plant proteins may be determined by skin-tests. Sterile watery extracts of such materials as horse-hair, feathers, various pollens, fish muscle, fruit, eggs, etc., are on the market in a form ready for use. A single line of scarification, about 1 cm. long, is made through a drop of fluid and various solutions may be tested at one time. Sensitivity to a particular extract is indicated by a reaction of the wheal and erythema type, which appears within 10 minutes and attains its greatest size in from 30 minutes to an hour. The examination of the responses to these skin tests in a patient suffering from hay fever or asthma occasionally provide information of value in the prevention of further attacks.

#### XIV SOME TESTS USED IN RHEUMATIC DISEASES

**Antistreptolysin test.** Haemolytic streptococci produce two soluble haemolysins, which can act as antigens. The serum of a person

who has had a recent infection with haemolytic streptococci may contain antistreptolysins, that is, antibodies to one of these haemolysins. The test measures the titre of such antibodies. Raised levels are found two to three weeks after a streptococcal sore throat or scarlet fever, and four to five weeks after the onset of an attack of rheumatic fever. Levels of up to 100 are occasionally found in persons without a history of recent infection. Levels of 200 or over may be found after known streptococcal infections and even higher ones in rheumatic fever: such levels are not diagnostic of rheumatic fever, but merely indicate a previous infection with haemolytic streptococci. The test is of value in a negative sense in that rheumatic fever can virtually be excluded if the level is 50 units or less.

**The Rose-Waaler test.** This test depends on the fact that sera from many cases of rheumatoid arthritis have the property of agglutinating to high-titre sheep cells sensitized with rabbit anti-sheep cell amboceptor. The test has undergone various modifications, but the principle remains the same.

The test is positive in a titre of 1 in 32 in over 70 per cent of cases of rheumatoid arthritis. It may also be positive in other conditions associated with hyperglobulinaemia such as lupus erythematosus, liver disease, and occasionally in virus infections, amyloid disease, sarcoidosis, Hodgkin's disease, and ulcerative colitis. It is negative, however, in other rheumatic diseases (acute rheumatism, gout, osteo-arthritis, etc.), and thus when positive may be valuable in the diagnosis of cases of monarticular or otherwise atypical rheumatoid arthritis. When negative, it does not exclude this condition.

**Latex fixation test.** This is a simple modification of the Rose-Waaler test in which polystyrene latex particles are sensitized to react with sera containing rheumatoid factor. It is usually reported as positive or negative with an indication of the intensity of the reaction. The results agree fairly closely but not completely with the original method, and the pitfalls in interpretation are similar.

## XV PREGNANCY TESTS

The urine of both normal men and women contains small amounts of the gonad-stimulating hormone, derived from the anterior lobe

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of the pituitary. During pregnancy, this hormone is also produced by the chorionic cells of the placenta and the amount in the urine is increased. It is greatly increased in certain abnormal conditions such as hydatidiform mole, chorioncarcinoma and teratoma of the testis. This is the basis of the Ascheim-Zondek and other pregnancy tests.

An early-morning specimen of urine from the patient is collected into a sterile bottle and is injected into immature female animals, either mice or rats. The animal reacts to the excess of hormone in a pregnancy-urine by erythema or haemorrhagic follicles in the ovary. The test may be made quantitative by using falling dilutions of the urine.

Slide tests for pregnancy are now commonly used instead of the older biological tests. There are two stages in these tests. An anti-serum can be prepared against human chorionic gonadotrophin and this can be neutralized by the excess hormone in pregnancy urine, if the two are mixed together. To detect such neutralization, latex particles coated with hormone are used. These remain in suspension if no antibody to the hormone is present, but are agglutinated if antibody is present. Thus in the test a drop of anti-serum is mixed with a drop of patient's urine, and to the mixture is added a drop of latex particles coated with hormone. If agglutination is seen, then the anti-serum has not been neutralized by the urine and the patient is not pregnant.

## XVI SIMPLE BIOCHEMICAL TESTS

**1 Serum bilirubin.** An excess of bilirubin in the blood may result from an increased rate of destruction of red cells (haemolysis), or from interference with excretion owing to liver-damage (intra-hepatic obstruction) or to obstruction of the bile-ducts (extra-hepatic obstruction). In both these forms of obstruction, bile which has passed through the liver cells may be reabsorbed into the blood-stream. Estimations of the serum bilirubin (normal up to 0.5 mg. per 100 ml.) are used to detect subclinical jaundice (0.5 to 2.0 mg. per 100 ml) when no abnormal coloration may be visible, and to follow the progress of cases of jaundice.

**2 The blood urea.** Normal blood contains from 20 to 40 mg. of urea per 100 ml. With moderate reduction of renal efficiency normal values are usually found. With more severe degrees of

Plate I



Cretinism



Mongolism



Cushing's syndrome



Tetanus

FACIES

Plate II



Myxoedema



Hyperthyroidism



Nephrotic oedema



Tabes dorsalis

FACIES

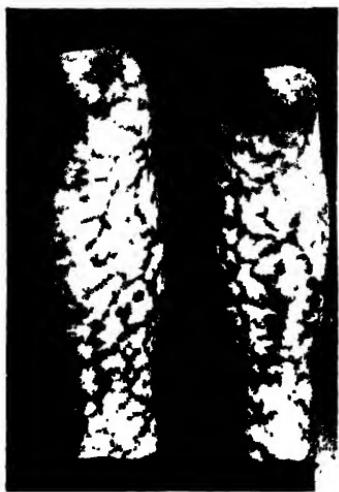
Plate III



Leucoderma



Ultraviolet irradiation



Livedo reticularis



Arsenic pigmentation.

PIGMENTATION OF SKIN

**Plate IV**



**Pellagra**



**Addison's disease and normal control**

**PIGMENTATION OF SKIN**

Plate V



Tetany



Gout



Rheumatoid arthritis



Heberden's nodes



Koilonychia



Clubbing

HAND

Plate VI



a

- a Black hairy tongue
- b Geographical tongue
- c False geographical tongue



b



TONGUES

Plate VII



- a Congenital fissures
- b Chronic superficial glossitis
- c Median rhomboid glossitis



TONGUES

Plate VIII



Occlusion of inferior vena cava



Venous anastomosis in portal  
obstruction



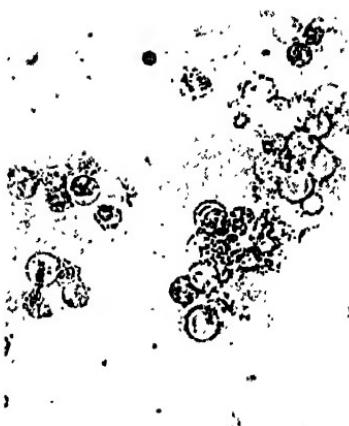
Arterial anastomosis on shoulders in  
coarctation of aorta

**COLLATERAL CIRCULATIONS**

Plate IX



Red cells (crenated) and  
epithelial cells  
( $\sim 200$  approx.)



Pus cells  
( $\sim 250$  approx.)



Granular cast  
( $\sim 150$  approx.)



Hyaline cast  
( $\sim 95$  approx.)

CELLS AND CASTS IN URINE

Plate X



Old rickets



Ankylosing  
spondylitis



Parkinsonism



Spastic diplegia

ATTITUDE

Plate XI



Hydrocephalus



Rickets



Osteitis Deformans



Acromegaly

SKULL

Plate XII

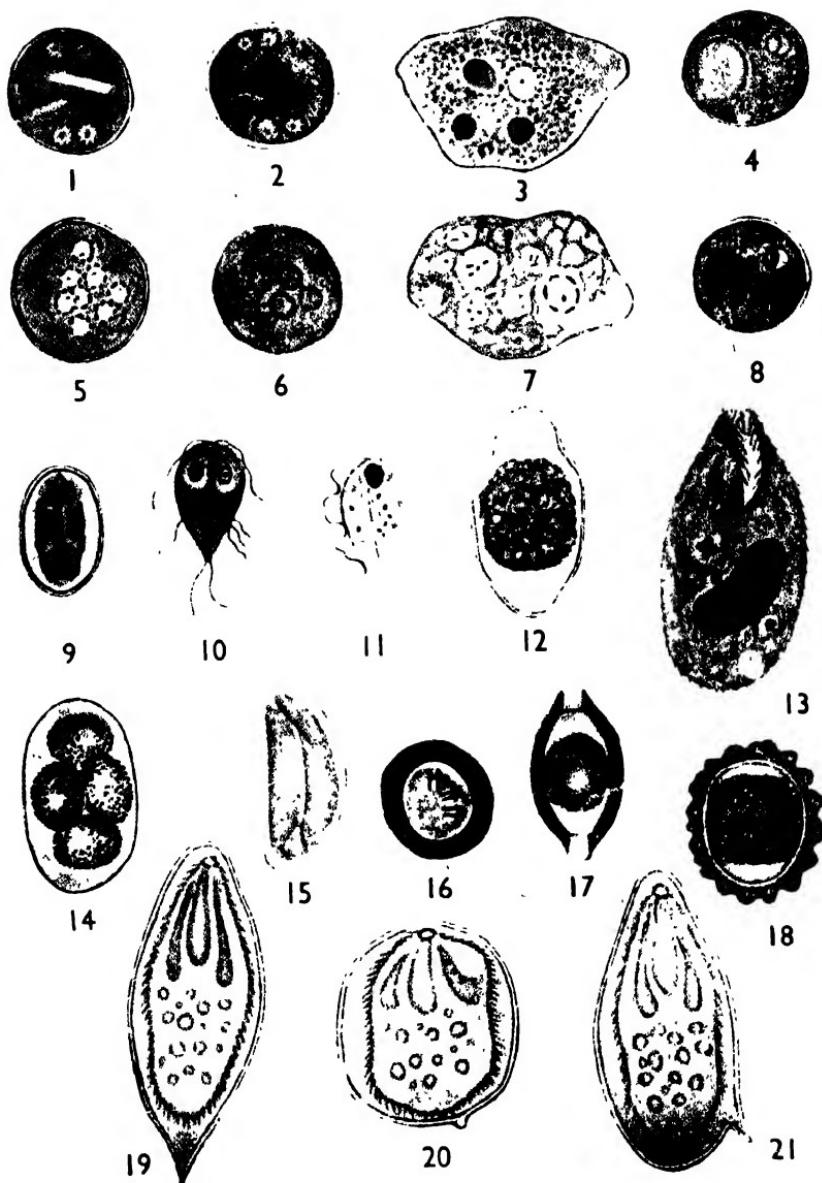
INTESTINAL PARASITES

- 1 *Entamoeba histolytica*. Fully developed 4-nucleated cyst containing chromatoid bodies, as seen in saline preparations (- 1500).
- 2 *Entamoeba histolytica*. Four-nucleated cyst as seen in iodine preparation (- 1500).
- 3 *Entamoeba histolytica*. Active form, containing included red blood cells as seen in saline preparations (- 1500).
- 4 *Iodamoeba butschlii*. Cyst, as seen in saline preparations. Note unstained glycogen vacuole (- 1500).
- 5 *Entamoeba coli*. Fully developed 8-nucleated cyst, as seen in saline preparations (- 1500).
- 6 *Entamoeba coli*. Eight-nucleated cyst stained by Lugol's iodine solution (- 1500).
- 7 *Entamoeba coli*. Active form, as seen in saline preparations (- 1500).
- 8 *Iodamoeba butschlii*. Cyst stained by Lugol's iodine solution (- 1500).
- 9 *Giardia lamblia*. Cyst form, stained by Heidenhain's haematoxylin (- 1500).
- 10 *Giardia lamblia*. Active form stained by Heidenhain's haematoxylin (- 1500).
- 11 *Trichomonas hominis*. Stained by Giemsa's method (- 1500).
- 12 *Isospora bellii* (*I. hominis*). Undeveloped oocyst as passed in human faeces (- 500).
- 13 *Balantidium coli*. Active form stained by Heidenhain's haematoxylin (- 350).
- 14 Ova of *Ankylostoma duodenale* (Hookworm) (- 500).
- 15 Ova of *Enterobius vermicularis* (Threadworm) (- 500).
- 16 Ova of *Taenia solium* and *saginata* (Tapeworms) (- 500).
- 17 Ova of *Trichuris trichura* (Whipworm) (- 650).
- 18 Ova of *Aegyptiacis lumbricoides* (Roundworm) (- 500).
- 19 Ova of *Bilharzia haematobium* (- 300).
- 20 Ova of *Bilharzia leporinum* (- 300).
- 21 Ova of *Bilharzia mansoni* (- 300).

All magnifications approximate.

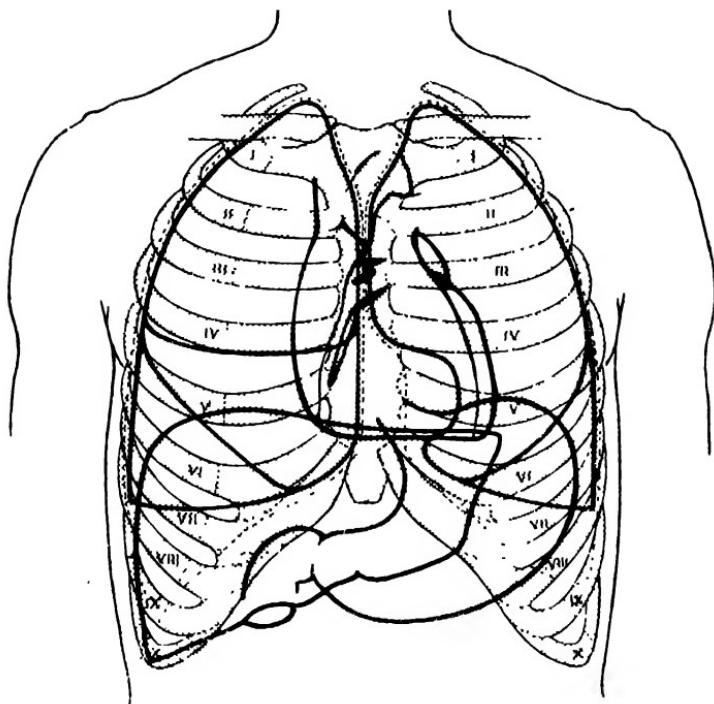
Drawn by W. Cooper.

Plate XII



INTESTINAL PARASITES

Plate XIII



VISCTRA OF THORAX AS SEEN FROM  
THE FRONT IN THE CADAVER

Legend to Plate XIV

*Plasmodium vivax* 1 Ring stage 2 Amoeboid form 3 Fully developed schizont 4 Male gametocyte 5 Female gametocyte.

*Plasmodium malariae* 6 'Compact' form 7 'Band' form. 8 Fully developed schizont.

*Plasmodium ovale* 9 Female gametocyte 10 Fully developed schizont

*Plasmodium falciparum* 11 Red blood corpuscles containing multiple infections of various types of young rings. 12 'Old' ring, showing altered staining reaction and Maurer's dots. 13 Male gametocyte or crescent. 14 Female gametocyte or crescent. 15 Pigment in polymorpho 'clear leucocyte'.

*Cryptosporidium donovani* (from a spleen smear) 16 Some lying free, others within the cytoplasm of an endothelial cell.

*Trypanosoma cruzi* 17 Adult form as seen occasionally in the blood of patients suffering from Chagas disease.

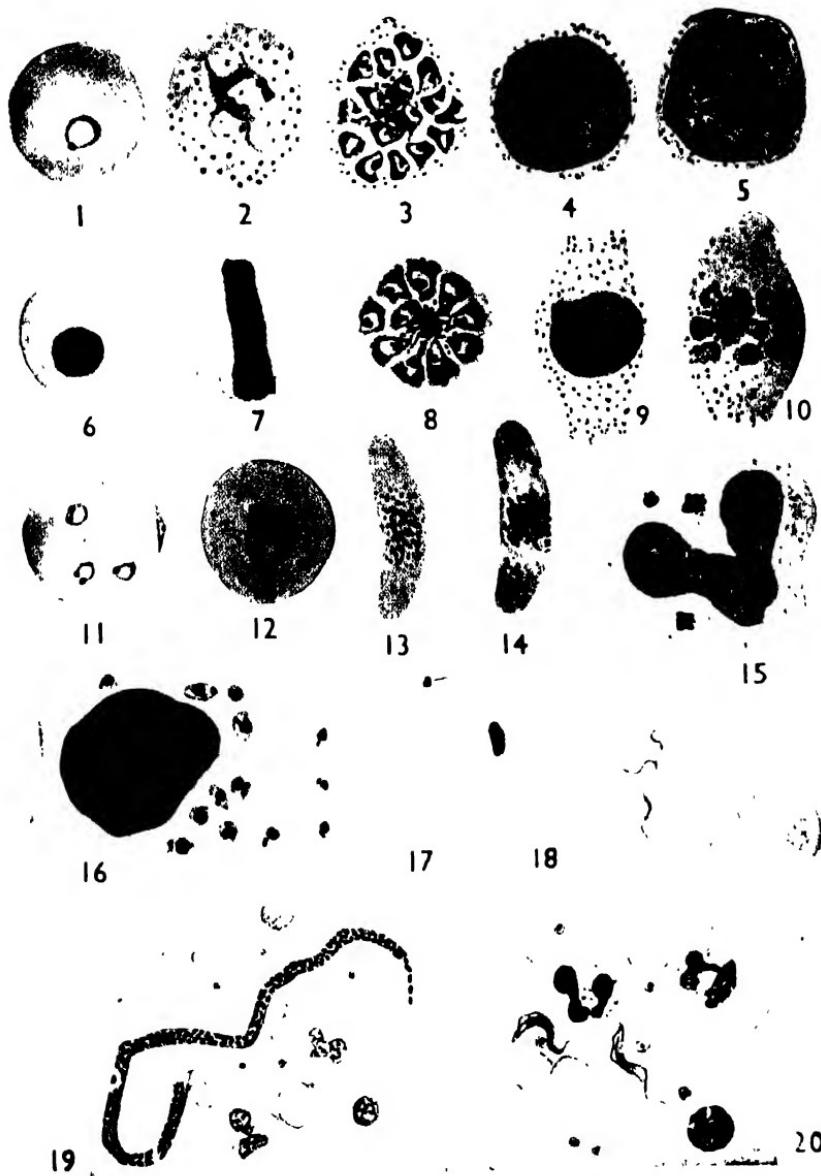
*Treponema recurvatum* 18.  
(Nos 1-18 magnification  $\times 2000$  approx.)

*Malaria itala loa loa* 19 ( $\times 600$  approx.).

*Trypanosoma rhodesiense* 20 As seen in a thick blood film of patients suffering from trypanosomiasis ( $\times 1000$  approx.).

Drawn by W. Cooper.

Plate XIV



PARASITES OF THE BLOOD

Plate XV



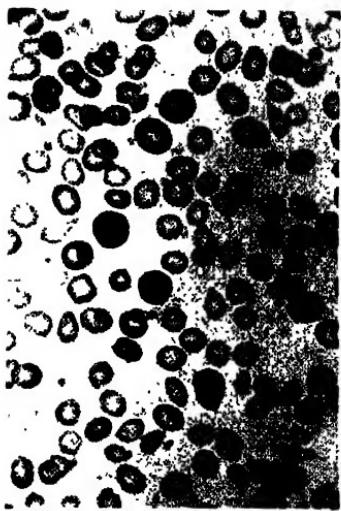
a Normoblasts

b, c Megaloblasts: The right showing early,  
intermediate, late and mitosis.



c

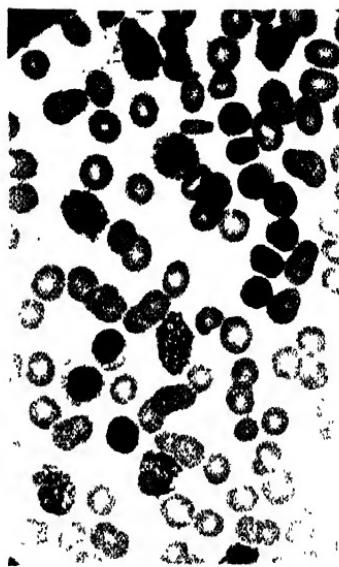
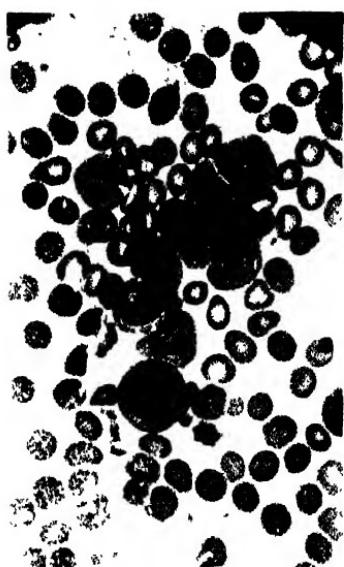
BONE MARROW CELLS



- a *Hypochromic Anæmia due to blood loss.* Note mixed population of hypochromic microcytes, some normochromic cells of normal size, and some polychromatic macrocytes.
- b *Pernicious Anæmia.* Note anisosytosis, poikilocytosis and a number of macrocytic cells.
- c *Sickle Cell Anæmia.* Numerous target cells present, and some sickle cells. Hypochromic microcytes also seen. Two normoblasts in the field.
- d *Hereditary Spherocytosis.* Some of the red cells are spherocytic—smaller diameter and deeply staining centre. Some polychromatic macrocytes.

THE ANAEMIAS

Plate XVII



- a *Chronic Myeloid Leukaemia*. Film shows mature polymorphs, band forms, metamyelocytes with indented nuclei, myelocytes with larger nuclei and granular cytoplasm, and one larger myeloblast containing nucleoli
- b *Chronic Lymphatic Leukaemia*. Large number of morphologically normal small lymphocytes, but bare nuclei also present.

LEUKAEMIA

Plate XVIII



a



b



c



d

- a *Bone Marrow in Myelomatosis* showing myeloma cells, some containing nucleoli, others with mature nuclei resembling normal adult plasma cells.
- b *Bone Marrow in Acute Leukaemia* showing blast cells with multiple nucleoli
- c *Peripheral Blood in Glandular Fever* showing large atypical mononuclear cells, the more deeply staining cell showing characteristic 'foamy' cytoplasm.
- d *Blood Cells in Acute Leukaemia, myelo-monocytic type* Note loose nuclear chromatin and nucleoli.

MISCELLANEOUS BLOOD CELL DISORDERS

Plate XIX



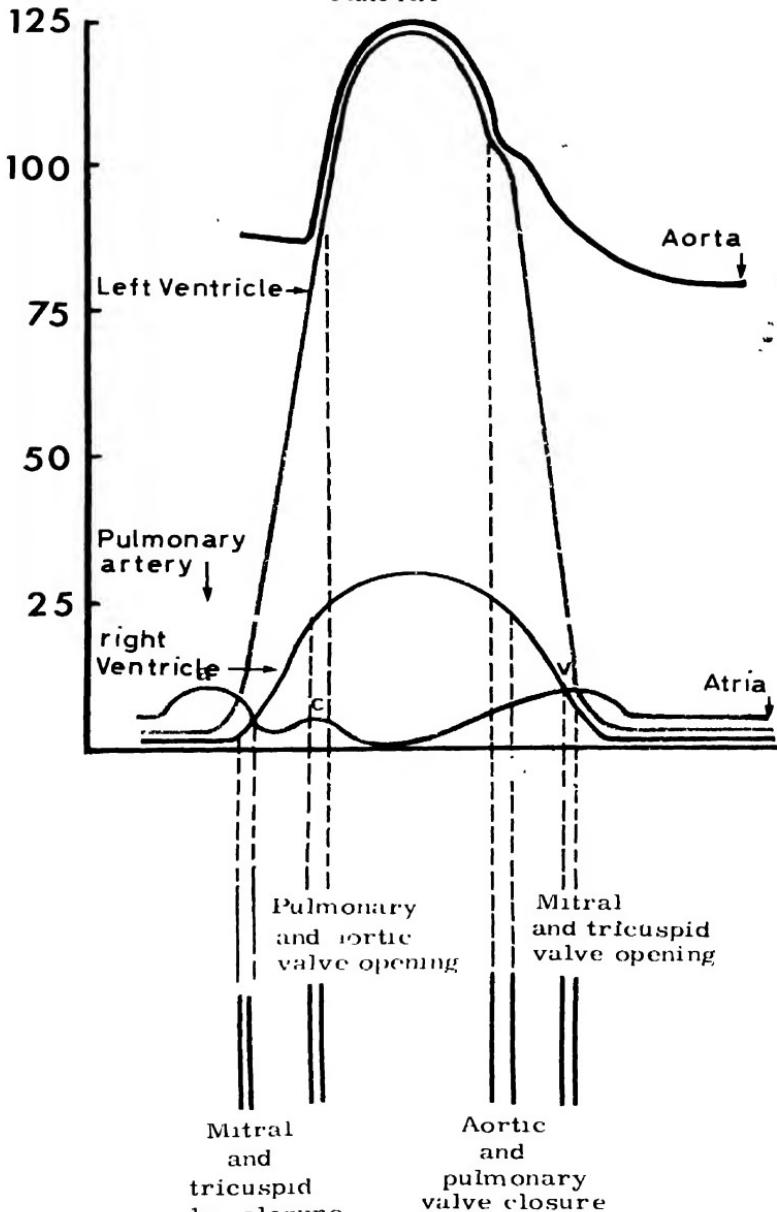
CARCINOMA CELLS IN THE SPUTUM



ASBESTOS BODIES IN THE SPUTUM

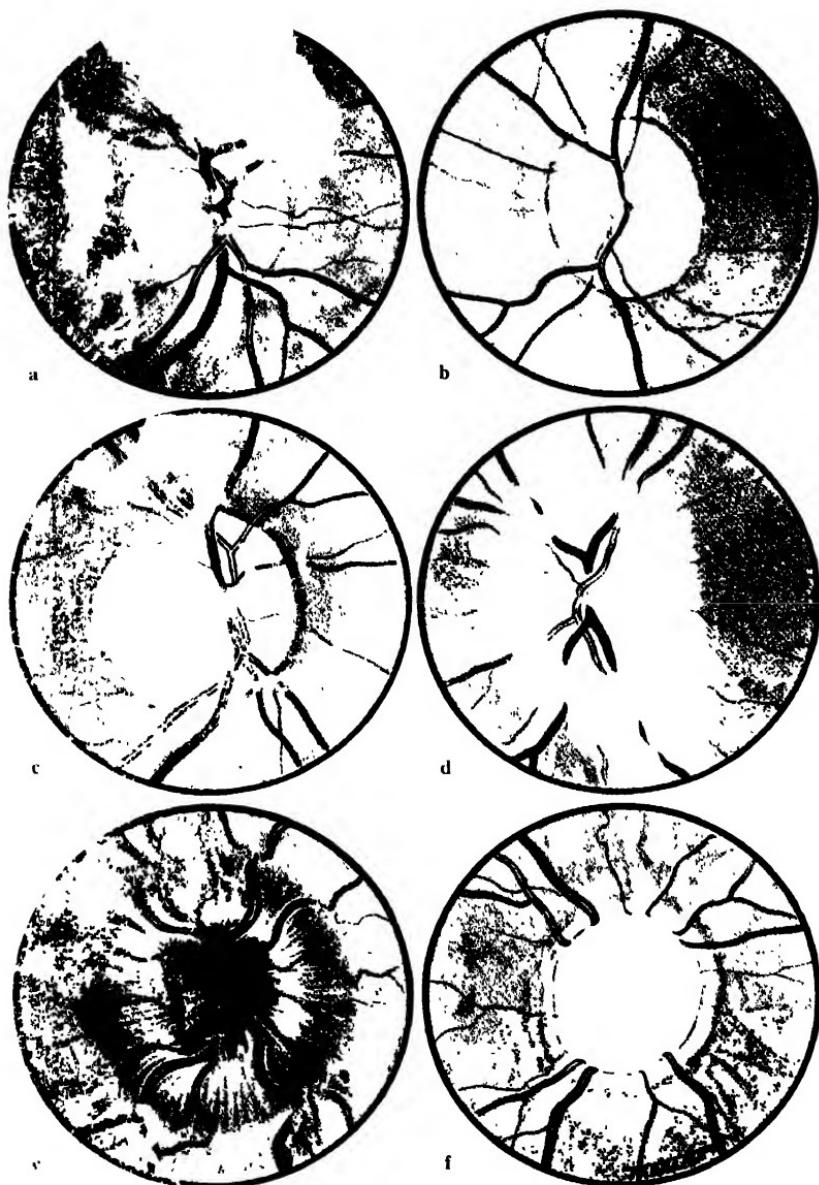
mmHG

Plate XX



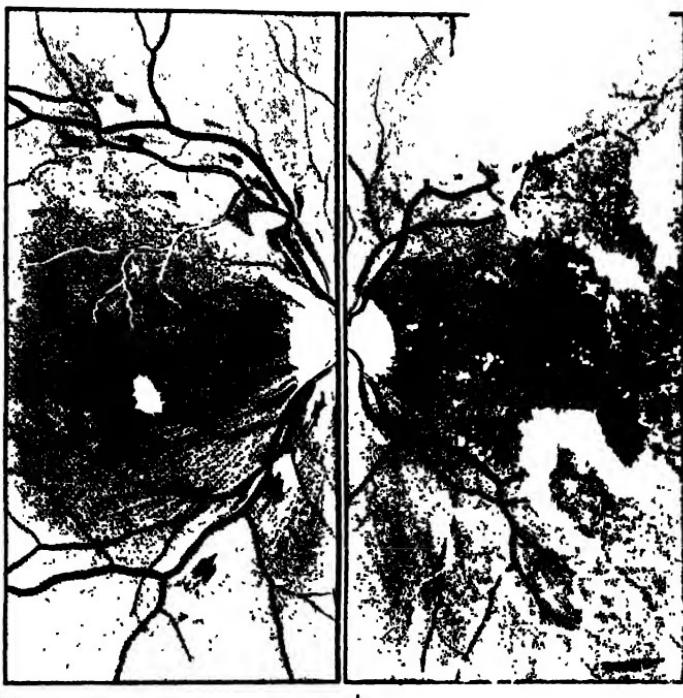
THE CARDIAC CYCLE

Plate XXI



FUNDUS OF THE EYE

Plate XXII



a *Hypertensive Retinopathy*. The arteries are irregular in calibre and show 'silver wire' arterio-venous nipping is present. Characteristic 'flame-shaped' haemorrhages & 'cotton-wool' exudates can be seen.

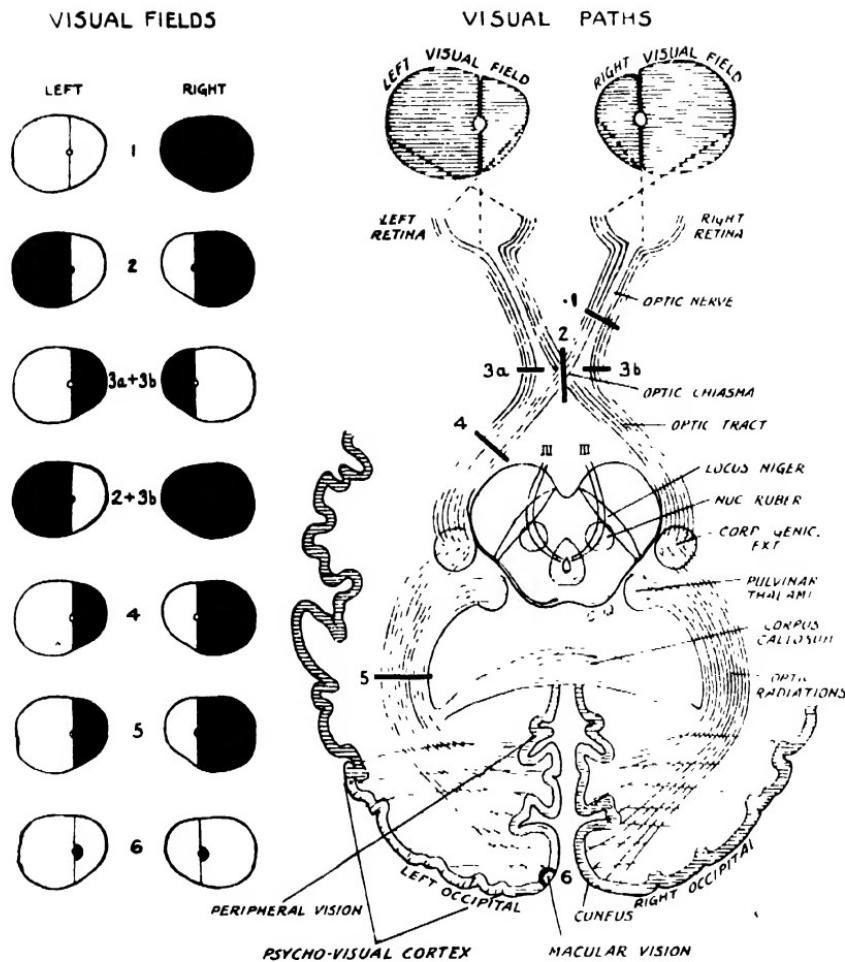
b *Diabetic Retinopathy*. Micro-aneurysms (tiny red dots), round haemorrhages, exudates and areas of new vessel formation are characteristic of this condition. In old patients with a diabetic retinopathy, a hypertensive retinopathy is also present.

## RETINOPATHY

### Legend to Plate XXI

- a *Normal Optic Disc*.
- b *Optic Atrophy*. The disc is white in colour and the number of small vessels crossing its edge is reduced.
- c *Myopic Crescent*. A crescent or ring of exposed white sclera seen particularly in high degrees of myopia.
- d *Opaque Nerve Fibres*. The characteristic feathery appearance of medullated nerve fibres.
- e *Papilloedema*. A red swollen optic disc with dilated veins. The retinal vessels bend sharply as they dip down from swollen disc to surrounding retina. A similar ophthalmoscopic appearance occurs in papillitis.
- f *Glaucomatous Cupping*. A form of optic atrophy due to raised intraocular pressure. The floor of the cup is depressed below the level of the surrounding retina.

Plate XXIII



- Lesion at 1 produces complete blindness in right eye, with loss of direct right reflex.
- " " 2 produces bitemporal hemianopia.
- " " 3a + 3b produces binasal hemianopia.
- " " 2 + 3b produces blindness of right eye, with temporal hemianopia of left visual field.
- " " 4 produces right homonymous hemianopia, with hemianopic pupillary response.
- " " 5 produces right homonymous hemianopia, with normal pupillary reaction to light.
- " " 6 produces right homonymous central hemisotoma.

(After Purves-Stewart)

reduction the blood-urea begins to rise, and may reach 600 mg. per 100 ml. before death. Patients with blood-urea above 100 mg. per 100 ml., not due to a temporary condition which can be relieved, rarely live more than a year. For methods of estimating the renal efficiency, see p. 184.

**3 The blood sugar.** The blood of normal persons, taken when they have fasted for some hours, contains 80 to 120 mg. of glucose per 100 ml. After a meal containing carbohydrate this may rise to 150 mg. Usually only traces of glucose appear in the urine when the blood-sugar is below 180 mg. per 100 ml., the 'renal threshold'.

In diabetes mellitus the blood may contain much larger quantities of glucose, up to 600. mg or more.

**4 The glucose-tolerance test.** To determine a patient's ability to metabolize carbohydrate, he is given a test dose of 50 g. of glucose, after which the blood sugar is estimated and the urine tested for the presence of sugar at intervals for the next two or three hours.

The usual method is as follows. The patient, who has been allowed no food since the previous evening, has blood taken for a fasting blood-sugar test and empties his bladder. He then drinks 50 g. of glucose dissolved in 100 ml. of water. Further specimens of blood are withdrawn and further samples of urine collected at the end of  $\frac{1}{2}$  hour and two hours (or after  $\frac{1}{2}$ , 1,  $1\frac{1}{2}$  and 2 hours),

Under normal conditions the fasting blood-sugar is between 80 and 120 mg. per 100 ml. The blood-sugar  $\frac{1}{2}$  hour after the glucose is taken has risen to 140 to 170 mg. per 100 ml., and at the end of two hours it has fallen to its fasting level. The corresponding specimens of urine contain no sugar, since sugar does not pass into the urine in detectable quantities in normal persons till the blood sugar reaches 180 mg. per 100 ml.—the so-called 'renal threshold'.

In renal glycosuria or 'lowered renal threshold' the blood-sugar curve is normal, but sugar is found in one or more of the corresponding specimens of urine.

This test is employed in the diagnosis of diabetes mellitus. For this purpose it is essential that the patient should have been eating a normal amount of carbohydrate during the previous week. Normal persons on a low carbohydrate diet may show abnormal blood sugar levels after a test dose of glucose, and hence be misdiagnosed as cases of mild diabetes, if this precaution is not

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observed. Cases of severe diabetes mellitus can generally be recognized by the history and examination of the urine, but if the glucose-tolerance test is performed the fasting blood-sugar is usually well above 120 mg. per 100 ml. and may be 200 or 300 mg. After the test dose of glucose the blood-sugar rises and does not return to normal levels at the end of two hours.

The significance of moderately raised blood-sugar curves without symptoms of diabetes is not known. Persons who have them sometimes progress to a true diabetes mellitus. If a single blood-sugar determination is used to confirm a diagnosis of diabetes mellitus, one taken an hour after a carbohydrate meal should be employed, rather than a fasting one. In true diabetes mellitus the blood-sugar at this time will be found to be 250 mg. per 100 ml. or more.

The glucose tolerance test finds its greatest usefulness, however, in excluding diabetes mellitus in persons with symptomless glycosuria.

## APPENDIX

### EQUIVALENTS: METRIC/IMPERIAL

#### Mass

1 kilogram (kg.)	= 15,432 grains or 35 274 ounces or 2.2046 pounds
1 grammme (G.)	= 15 432 grains
1 milligram (mg.)	= 0.015432 grain
—	—
1 pound (avoirdupois) (lb.)	= 453.59 grammes
1 ounce (avoirdupois) (oz.)	= 28.35 grammes
1 grain (gr.)	= 64.799 milligrams

#### Capacity

1 litre (l.)	= 1.7598 pints or 0.22 gallons
1 millilitre (ml.)	= 16.894 minims
—	—
1 pint (O or pt.)	= 568.25 millilitres or 0.56825 litre
1 fluid ounce (or fl. oz.)	= 28.412 millilitres
1 fluid drachm (or fl. dr.)	= 3.5515 millilitres
1 minim (M. or min.)	= 0.059192 millilitres

### APPROXIMATE EQUIVALENT DOSES IN THE METRIC AND IMPERIAL (APOTHECARIES') SYSTEMS

These tables may be used for the direct transference of doses from one system to the other. Multiples of these equivalents must not be used, since in multiplying, the error of the approximation might be raised to a significant figure.

#### Weights

Grammes	Grains	Milligrams	Grains
10	150	45	3
8	120	35	2
6	90	30	1
5	75	25	1.5
4	60	20	1.3
3	45	15	1.4
2.6	40	12	1.5
2	30	10	1.6

## APPENDIX

<i>Grammes</i>	<i>Grains</i>	<i>Milligrams</i>	<i>Grains</i>
1.6	25	8	$\frac{1}{8}$
1.3	20	6	$\frac{1}{10}$
1	15	5	$\frac{1}{2}$
0.8	12	4	$\frac{1}{6}$
0.6	10	3	$\frac{1}{20}$
0.5	8	2.5	$\frac{1}{24}$
		2	$\frac{1}{30}$
		1.5	$\frac{1}{40}$
<i>Milligrams</i>	<i>Grains</i>	<i>1.2</i>	$\frac{1}{50}$
400	7	1	$\frac{1}{60}$
300	5	0.8	$\frac{1}{80}$
250	4	0.6	$\frac{1}{100}$
200	$3\frac{1}{2}$	0.5	$\frac{1}{120}$
150	$2\frac{1}{2}$	0.4	$\frac{1}{160}$
120	2	0.3	$\frac{1}{200}$
100	$1\frac{3}{4}$	0.25	$\frac{1}{240}$
80	$1\frac{1}{3}$	0.2	$\frac{1}{300}$
75	$1\frac{1}{4}$	0.15	$\frac{1}{400}$
60	1	0.12	$\frac{1}{500}$

## Fluid Measures

<i>Millilitres</i>	<i>Minims</i>	<i>Millilitres</i>	<i>Minims</i>
10	150	0.8	12
8	120	0.6	10
6	90	0.5	8
5	75	0.4	6
4	60	0.3	5
3	45	0.25	4
2.6	40	0.2	3
2	30	0.15	$2\frac{1}{2}$
1.6	25	0.12	2
1.3	20	0.1	$1\frac{1}{2}$
1	15		

1 fluid ounce = approx. 30 ml.

1 fluid drachm = approx. 4 ml.

15 minims = approx. 1 ml.

## Length

1 metre (m.)	= 39.370 inches
1 decimetre (dm.)	= 3.9370 inches
1 centimetre (cm.)	= 0.39370 inch
1 millimetre (mm.)	= 0.039370 inch
1 micron( $\mu$ )	= 0.000039370 inch

1 inch = 25.400 millimetres

## 3 Conversions.

To convert grammes per 100 ml. into grains per ounce, multiply by 4.375.

To convert grammes into ounces avoirdupois, multiply by 10 and divide by 283.

To convert litres into pints, multiply by 88 and divide by 50.

To convert kilos into pounds, multiply by 1000 and divide by 454.

#### 4 Centigrade and Fahrenheit scales

To convert Fahrenheit into Centigrade, subtract 32, multiply the remainder by 5, and divide the result by 9.

To convert Centigrade into Fahrenheit, multiply by 9, divide by 5, and add 32.

The following table and figure show the relation of degrees Fahrenheit to Centigrade, as far as is likely to be required in clinical work.

F	C	Centigrade	Fahrenheit	Centigrade	Fahrenheit
107	42	110°	. . . . .	230°	. . . . .
106	41	100.	. . . . .	212	. . . . .
		95.	. . . . .	203	. . . . .
		90.	. . . . .	194	. . . . .
	40	85.	. . . . .	185	. . . . .
		80.	. . . . .	176	. . . . .
		75.	. . . . .	167	. . . . .
103	39	70.	. . . . .	158	. . . . .
102	39	65.	. . . . .	149	. . . . .
		60.	. . . . .	140	. . . . .
		55.	. . . . .	131	. . . . .
101	38	50.	. . . . .	122	. . . . .
100	38	45.	. . . . .	113	. . . . .
		44.	. . . . .	111·2	. . . . .
99	37	43.	. . . . .	109·4	. . . . .
		42.	. . . . .	107·6	. . . . .
98	37	41.	. . . . .	105·8	. . . . .
		40·5	. . . . .	104·9	. . . . .
97	36	40.	. . . . .	104·0	. . . . .
		39·5	. . . . .	103·1	. . . . .
96	35	39.	. . . . .	102·2	. . . . .
		38·5	. . . . .	101·3	. . . . .
		38.	. . . . .	100·4	. . . . .
		37·5	. . . . .	99·5	. . . . .
		37°	. . . . .	98·6°	. . . . .

Fig. 80  
Fahrenheit  
and Centigrade  
scales compared.

#### 5 Some normal values

There are unfortunately no universally agreed standards of normality or limits of normality. The figures given here are believed to be those usually accepted in Great Britain. But different laboratories have different methods and different standards, and in case of doubt the laboratory performing the investigations should always be consulted about the interpretation of the result.

Haematological investigations (average and limits of normal stated where possible)						
Red cells per mm. <sup>3</sup>	.	.	.	.	.	Adult males 5,500,000 5,000,000 to 6,500,000
						Adult females 4,800,000 4,000,000 to 5,500,000
White cells per mm. <sup>3</sup>	.	.	.	.	.	7,000 3,000 to 12,000
Eosinophils per mm. <sup>3</sup>	.	.	.	.	.	150 to 400
Platelets per mm. <sup>3</sup>	.	.	.	.	.	150,000 to 500,000
Haemoglobin in g. per 100 ml. (100% = 14.6 g. per 100 ml.)	.	.	.	Adult males 13.5-18 g. per 100 ml. 92% to 123%		
				Adult females 11.5-16.4 g. per 100 ml. 79% to 112%		
Packed cell volume (PCV) per cent	.	.	.	.	.	Males 47 (42 to 50) Females 42 (38 to 45)
Mean corpuscular volume (MCV) in cubic microns ( $\mu^3$ )	.	.	.	.	.	86 (78 to 94)
† Mean corpuscular haemoglobin (MCH) in picograms (pg)	.	.	.	.	.	39.5 27 to 32
Mean corpuscular haemoglobin concentration (MCHC) per cent	.	.	.	.	.	34 32 to 38
Mean corpuscular diameter (MCD) in microns ( $\mu$ )	.	.	.	.	.	7.2 6.7 to 7.7
Reticulocytes per cent	.	.	.	.	.	0.5 to 2
Osmotic fragility of red cells	.	.	.	.	.	Haemolysis begins at 0.48 per cent saline and is complete at 0.32 per cent saline
Coagulation time:						
Lec and White	.	.	.	.	.	up to 3 mins.
Dale and Laidlaw	.	.	.	.	.	5 to 11 mins.
Bleeding time	.	.	.	.	.	2 to 7 mins.
Prothrombin time	.	.	.	.	.	12 to 14 seconds
Sedimentation rate:						
Westergren	.	.	.	.	.	Men 3-5 mm. in 1 hour Women 4-7 mm. in 1 hour
Wintrobe	.	.	.	.	.	Men 0-9 mm. in 1 hour Women 0-20 mm. in 1 hour
Red Cell Volume	.	.	.	.	.	Males 28-35 ml./kg. Females 23-30 ml./kg.
Plasma Volume	.	.	.	.	.	40-50 ml./kg.
Total Blood Volume	.	.	.	.	.	65-85 ml./kg.
Serum Iron	.	.	.	.	.	75-175 $\mu$ g/100 ml.
Serum Iron Binding Capacity	.	.	.	.	.	250-350 $\mu$ g/100 ml.
Serum Folate ( <i>L. Casei</i> )	.	.	.	.	.	3-10 ng/1 ml.*†
Serum B <sub>12</sub> ( <i>L. Leishmannii</i> )	.	.	.	.	.	150-1000 pg/1 ml.*†

\* Normals vary from individual laboratories.

† Note

1 milligram (mg) =  $\frac{1}{1000}$  of a gram      1 nanogram (ng) =  $\frac{1}{1000000000}$  of a gram

1 microgram (ug) =  $\frac{1}{1000000}$  of a gram      1 picogram (pg) =  $\frac{1}{1000000000000}$   
of a gram

**Blood chemistry (per 100 ml. of serum or plasma unless otherwise stated)**

Bicarbonate . . . . .	26-32 mEq/l
Calcium . . . . .	8·6-11·0 mg. per 100 ml.
Chloride . . . . .	95-110 mEq/l
Cholesterol . . . . .	150-240 mg. per 100 ml.
Total Lipids . . . . .	450-850 mg./100 ml.
Glucose . . . . .	80-150 mg. per 100 ml.
<b>Phosphatases:</b>	
Acid . . . . .	0-5 King-Armstrong units
Alkaline . . . . .	3-12 King-Armstrong units
Phosphorus . . . . .	2·5-4·5 mg. per 100 ml.
Potassium . . . . .	3·5-5·5 mEq/l
<b>Protein:</b>	
Albumin . . . . .	4·0-5·5 g. per 100 ml.
Globulin . . . . .	1·4-3·0 g. per 100 ml.
Albumin/globulin ratio . . . . .	3·1-1·5·1
Sodium . . . . .	135-147 mEq/l
<b>Transaminases:</b>	
SGOT . . . . .	5-40 units/ml. of serum
SGPT . . . . .	5-35 units/ml. of serum
Urea . . . . .	20-40 mg. per 100 ml.
Uric acid . . . . .	2-4 mg. per 100 ml.
Magnesium . . . . .	1·2-1·8 m Eq./l.
Carotene . . . . .	50-200 µg/100 ml.
Cortisol . . . . .	midnight 5-15 µg/100 ml. morning 10-25 µg/100 ml.
PBI . . . . .	3·5-7·5 µg/100 ml.
pH . . . . .	7·38-7·42
Po <sub>2</sub> . . . . .	37-45 mm. Hg
Pco <sub>2</sub> . . . . .	75-100 mm. Hg

**Liver-function tests**

Serum bilirubin . . . . .	0-0·5 mg. per 100 ml.
Alkaline phosphatase . . . . .	3-12 King-Armstrong units per 100 ml. (adults) 10-30 King-Armstrong units per 100 ml. (children)
Thymol turbidity . . . . .	0-4 units
Zinc sulphate turbidity . . . . .	0-8 units
Plasma proteins } see above	
Transaminases . . . . .	

**Urine chemistry**

Calcium . . . . .	up to 200 mg./24 hours
Chlorides . . . . .	5-15 g./24 hours
17 Oxosteroids:	
Adult males . . . . .	10-20 mg./24 hours
Adult females . . . . .	5-20 mg./24 hours
17 Oxogenic steroids:	
Adult males . . . . .	10-19 mg./24 hours
Adult females . . . . .	5-13 mg./24 hours

Catechol amines . . . . .	up to 150 mg./24 hours
VMA . . . . .	1-8 mg./24 hours
5-hydroxy-indole acetic acid . . . . .	3-10 mg./24 hours
Urea . . . . .	15-35 g./24 hours
Urobilinogen . . . . .	up to 4 mg./24 hours
<b>Faeces</b>	
Urobilinogen . . . . .	up to 300 mg./24 hours
Fat . . . . .	up to 6 g./24 hours
Calcium . . . . .	up to 610 mg./24 hours
<b>Cerebro-spinal fluid</b>	
Pressure . . . . .	60-150 mm. of fluid
Cells . . . . .	0-5 per mm. <sup>3</sup>
Protein . . . . .	20-40 mg. per 100 ml.
Glucose . . . . .	50-75 mg. per 100 ml.
Chlorides . . . . .	720-750 mg. per 100 ml.

## 6 Height and weight standard charts

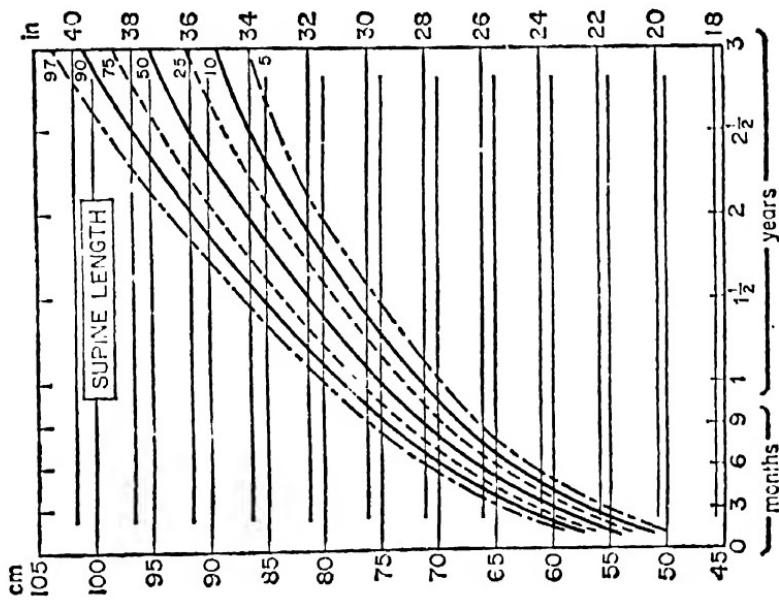
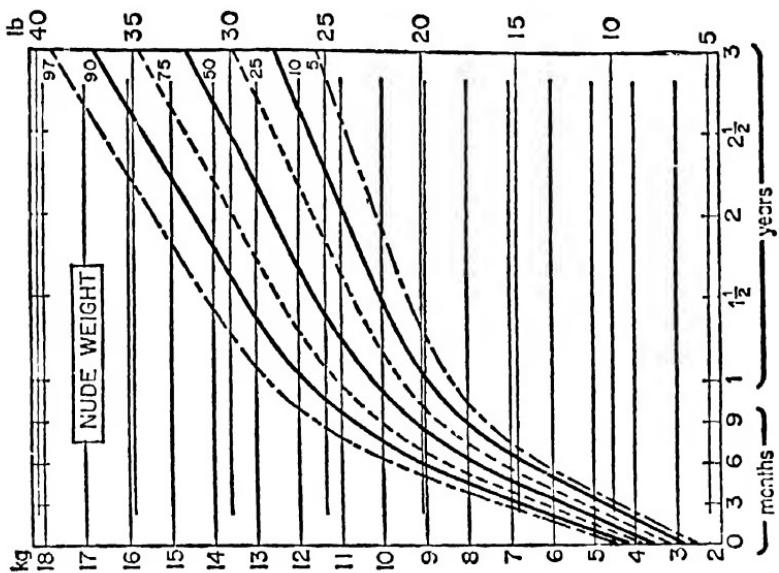
The charts on pp. 381-386 show standard heights and weights for boys and girls ages 0-3, 2-10, and 9-18. They have been modified from charts developed at the Institute of Child Health and are used by kind permission of J. M. Tanner and R. H. Whitehouse, Institute of Child Health and Hospital for Sick Children, Great Ormond Street, London. The original cards are obtainable from Joseph Collard & Sons, 48 Langham Street, London, W1. They include a page on which a child's growth and development can be recorded, and full directions for correct methods of measurement, etc., are also provided on each card.

The range of normal is expressed in percentiles and each chart shows the third, tenth, twenty-fifth, fiftieth, seventy-fifth, ninetieth, and ninety-seventh percentile. The meaning of the tenth percentile for height is that 10 per cent of all normal children are shorter than this height at the age concerned.

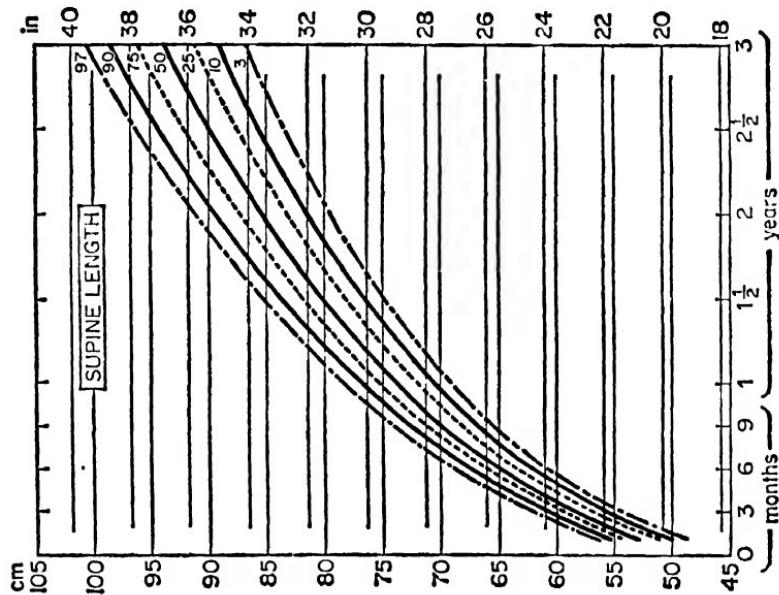
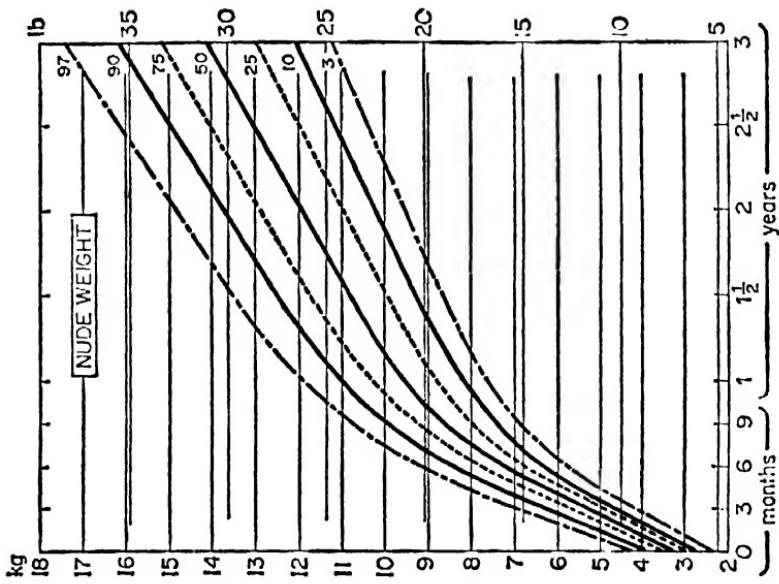
The limits of normality to be accepted must depend on the purpose for which the charts are being used and on local conditions. As a rough guide, however, it can be said that children outside the area of the tenth- to the ninetieth-percentile range should be regarded with slight suspicion, and those outside the third- to the ninety-seventh-percentile range as unhealthy unless proved otherwise.

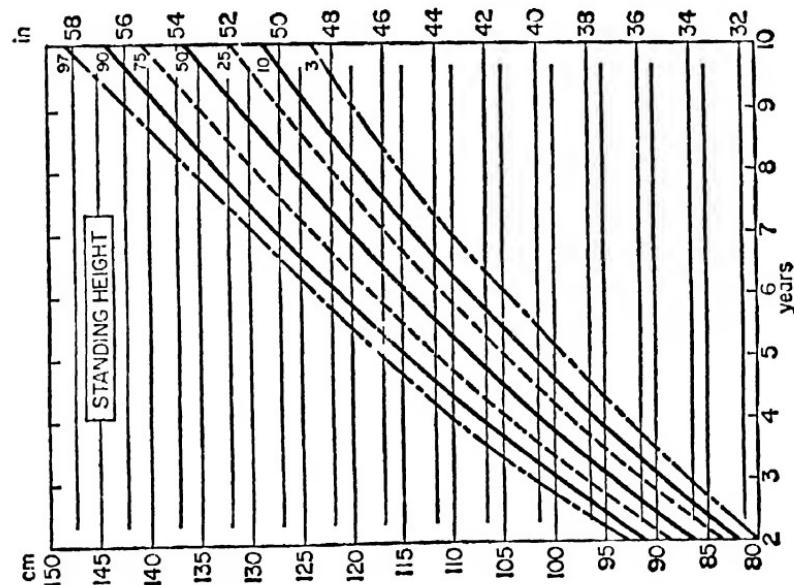
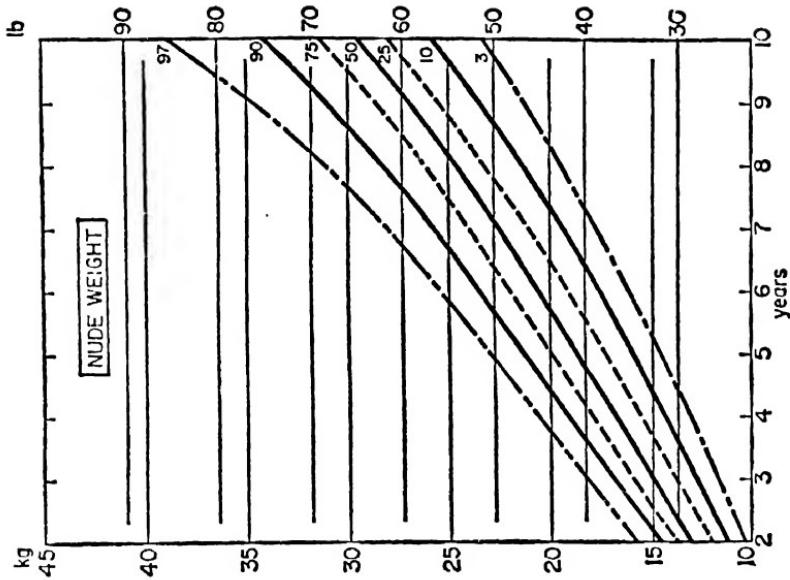
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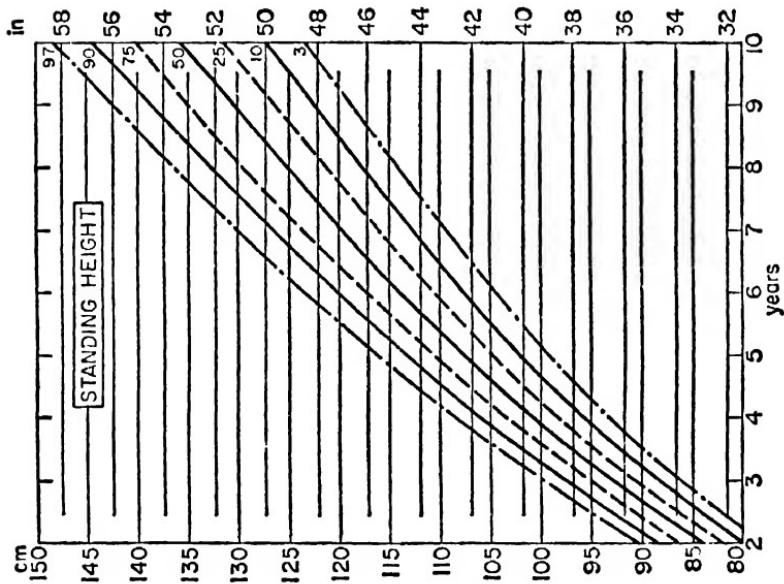
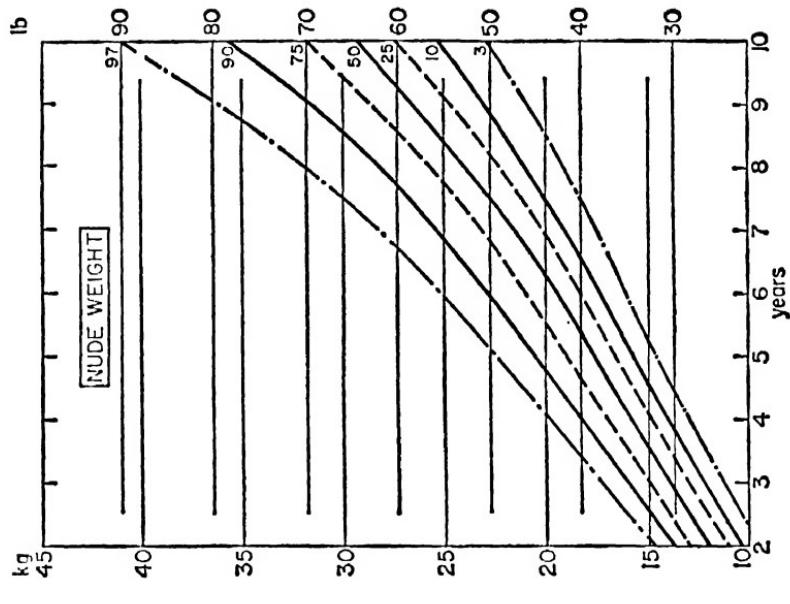


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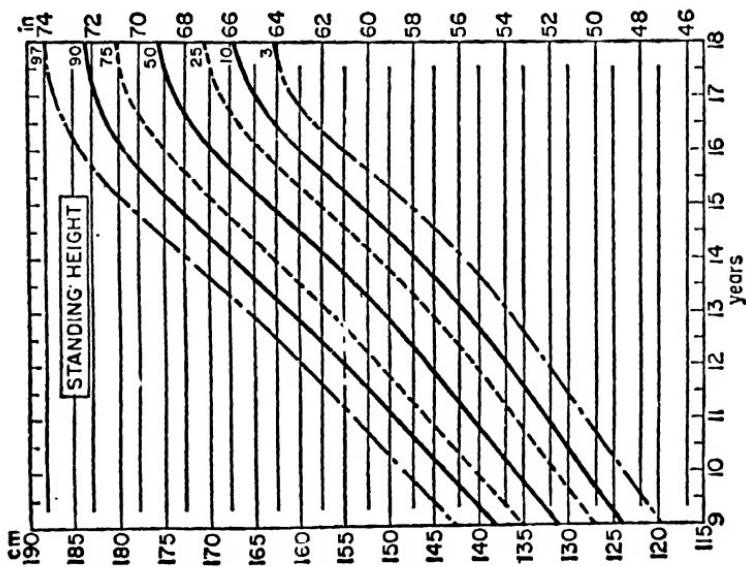
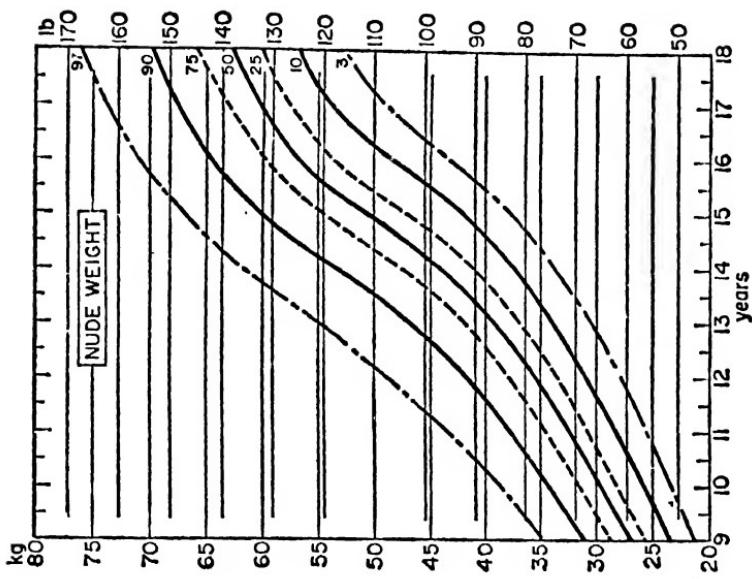


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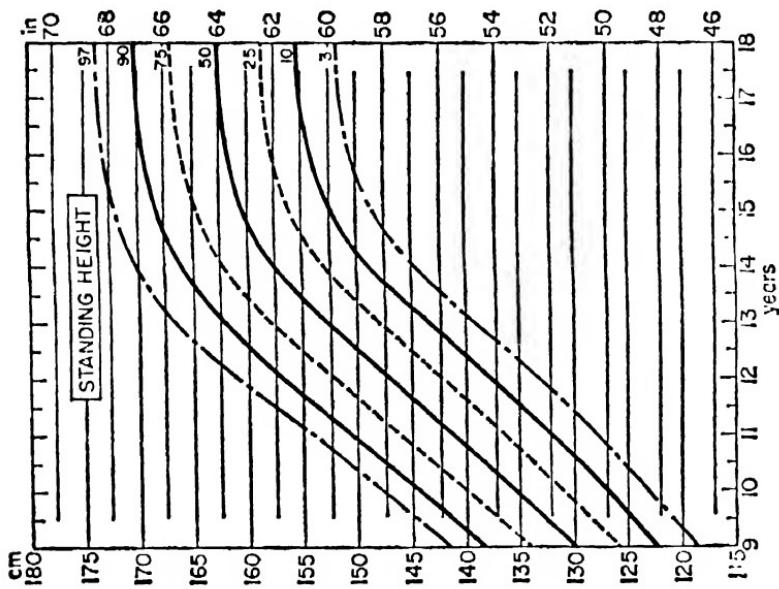
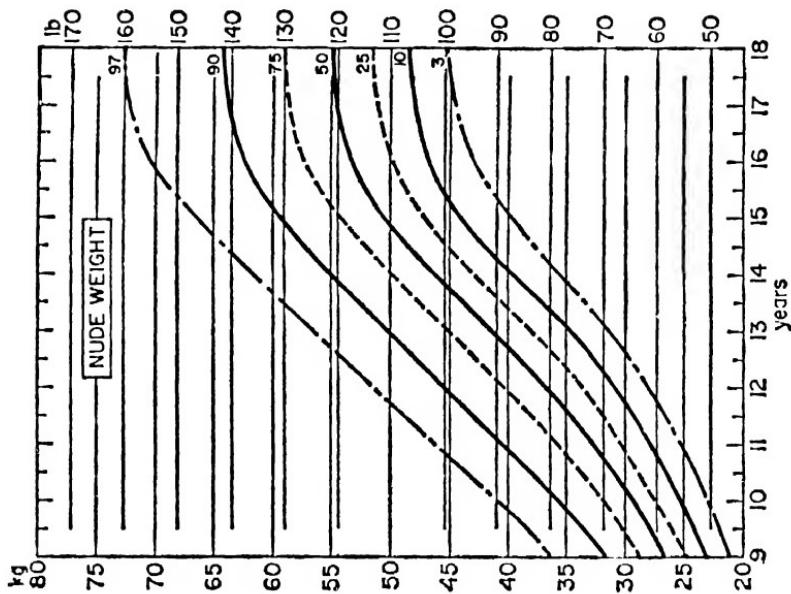


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